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Executive Dysfunction or State Regulation: A Dimensional Comparison of Two Neuropsychological Theories of Attention Disorder Symptoms Using RDoC Paradigms

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EXECUTIVE DYSFUNCTION OR STATE REGULATION: A DIMENSIONAL COMPARISON OF TWO NEUROPSYCHOLOGICAL THEORIES OF ATTENTION DISORDER SYMPTOMS USING RDOC PARADIGMS

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ABSTRACT

Two leading theories regarding the neurocognitive basis of attentional disorders are the executive dysfunction theory and the state regulation theory. The executive dysfunction theory takes a top-down approach, explaining the symptoms of ADHD as a byproduct of general deficits in executive functioning—particularly disinhibition. The state regulation theory takes a bottom-up approach, explaining the symptoms of ADHD as a failure to be sufficiently aroused by, and subsequently engage with, less stimulating or rewarding tasks. These two theories predict different patterns of performance on tasks of executive functioning and attention, and research has demonstrated mixed support for both theories. The present study used a continuous performance task to manipulate RDoC paradigms of inhibition and arousal predicted to be affected disparately according to each theory. The data failed to support either the executive dysfunction theory or the state regulation theory as hypothesized; however, there was a significant interaction between the paradigms used for each theory. Factor analysis may be useful in establishing predictions which may guide follow-up investigation.

CHAPTER 1

INTRODUCTION

The syndrome now called Attention-Deficit/Hyperactivity Disorder, or ADHD, was first described medically in 1775 by the German physician Melchior Adam Weikard (Barkley & Peters, 2012). Prior to the publication of the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III), this syndrome was grouped with other disorders, such as dyslexia, under a variety of diagnostic terms which reflected the underlying assumption of a neurological basis for the condition, including minimal brain dysfunction (Schmitt, 1975), and hyperkinetic reaction of childhood (American Psychiatric Association, 1968). With the DSM-III, the term Attention-Deficit Disorder (ADD) was coined, and specifiers for with or without hyperactivity were provided (APA, 1980). The condition has been officially called ADHD since DSM-III-R (APA, 1987), with the current subtypes of ADHD, predominantly inattentive type (ADHD-I); ADHD, predominantly hyperactive type (ADHD-H); and ADHD, combined type (ADHD-C) presented starting with DSM-IV (APA, 1994).

Is ADHD One Disorder?

Research to establish the DSM-IV subtypes of ADHD as distinct pathology or subtypes of a common disorder has been inconsistent over the past 20 years. Faraone, Biederman, Weber, and Russell (1998) did not find differences in cognitive or psychosocial functioning between ADHD subtypes, but noted that individuals diagnosed with ADHD-C were more likely to have comorbid psychiatric problems. Similarly, Geurts, Verte, Oosterlaan, Roeyers, and Sergeant (2005) found that ADHD subtypes (combined vs. inattentive) did not differ from controls on four out of five executive functioning tasks which are believed to access the primary neurocognitive

deficit in ADHD, although children with ADHD-C did differ from the control group on tasks related to inhibition.

On the other hand, several studies have identified ADHD-I as a distinct disorder from ADHD with hyperactivity (Barkley, DuPaul, and McMurry, 1990). Many of these studies identified a pattern of "sluggish cognitive tempo" (SCT) which can discriminate between ADHD-I and ADHD-C (Lockwood, Marcotte, & Stern, 2001; Milich, Balentine, & Lynam, 2001). However, Carlson and Mann (2002) found that among individuals diagnosed with ADHD-I, sluggish cognitive tempo predicts a different pattern of impairment and more psychiatric disorders compared to ADHD-I without sluggish cognitive tempo. This may indicate that ADHD-I consists of at least two distinct disorders—ADHD-I without SCT, sharing an etiology with the ADHD-C subtype, and ADHD-I with SCT, belonging to a separate etiology. At least one study (Graham et al., 2013) identified SCT as a core neurocognitive symptom of Fetal Alcohol Syndrome (FAS), and the authors were able to use SCT to differentiate between FAS and ADHD-I. This could indicate that FAS, which shares executive dysfunction as a core symptom with ADHD, may overlap considerably with ADHD-I in milder cases.

Heterogeneity of symptoms: The Research Domain Criteria (RDoC).

In 2013, the National Institute of Mental Health (NIMH) published its Research Domain Criteria (RDoC) for mental health research. Unsatisfied with the biological heterogeneity of mental disorders as classified under the taxonomy presented in the most recent update of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), NIMH published a matrix which identifies symptoms, rather than categorical disorders, as the focus of funded mental health research. This decision was a consequence of decades of heterogeneous findings in

genetic and physiological studies which indicate that the traditional presence/absence diagnostic taxonomy continued in the DSM-5 does not reflect the underlying biology of mental illness. The stated goal of RDoC is not to replace the DSM, but to provide a research-oriented foundation to facilitate the alignment of future mental health diagnostic systems with the biological basis of the symptoms (Cuthbert & Insel, 2010).

The RDoC matrix lists different systems (i.e., negative valence systems, positive valence systems, cognitive systems, social processes, and arousal and regulatory systems), and constructs (e.g., language, cognitive control) and subconstructs (e.g., response selection, inhibition/suppression, performance monitoring) within each system. For each construct/ subconstruct, RDoC list s number of elements within different units of analysis (i.g., genes, molecules, cells, circuits, physiology, behaviors, self-reports, and experimental paradigms).

While such an integrative approach is not entirely new to ADHD research (e.g., Waldman, 2005), the recommendation from NIMH to focus on the symptom-level is appropriate in light of the inconsistent results of research into the neurobiological model of ADHD. Even ignoring the developmental changes in neurochemistry and neurophysiology, as well as changes in behavior and cognitive functioning across the lifespan and between sexes, the diagnostic criteria (i.e., must have 6 of 9 symptoms of either inattention or hyperactivity/impulsivity, Fig. 1) create the possibility of 84 different combinations of symptoms for someone with a "pure" ADHD-predominantly Inattentive, and thousands of combinations resulting in ADHD-Combined type. The consequential heterogeneity of experience labeled as ADHD may be one reason for the heterogeneity of research outcomes in the literature.

Inattentive symptoms of ADHD

a. Often fails to give close attention in to details or makes careless mistakes in schoolwork, at work, or during other activities (e.g., overlooks or misses details, work is inaccurate).
b. Often has difficulty sustaining attention in tasks or play activities (e.g., has difficulty remaining focused during lectures, conversations, or lengthy reading)
c. Often does not seem to listen when spoken to directly (e.g., mind seems elsewhere, even in the absence of any obvious distraction).
d. Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., starts tasks but quickly loses focus and is easily sidetracked).
e. Often has difficulty organizing tasks and activities (e.g., difficulty managing sequential tasks; difficulty keeping materials and belongings in order; messy, disorganized work; has poor time management; fails to meet deadlines).
f. Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (e.g., Schoolwork or homework; for older adolescents and adults, preparing reports, completing forms, reviewing lengthy papers).
g. Often loses things necessary for tasks or activities (e.g., school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, and mobile phones).
h. Is often distracted by extraneous stimuli (for older adolescents and adults, may include unrelated thoughts).
i. Is often forgetful in daily activities (e.g., doing chores, running errands; for older adolescents and adults, returning calls, paying bills, keeping appointments).

Hyperactive/Impulsive symptoms of ADHD

a. Often fidgets with or taps hands or feet and squirm in seat.
b. Often leaves seat in situations when remaining seated is expected (e.g., leaves his or her place in the classroom, in the office or other workplace, or in other situations that require remaining in place).
c. Often runs about or climbs in situations where it is inappropriate. (Note: In adolescents or adults, may be limited to feeling restless).
d. Often unable to play or engage in leisure activities quietly.
e. Is often "on the go acting as if "driven by a motor" (e.g., is unable to be or uncomfortable being still for extended time, as in restaurants, meetings; difficult to keep up with).
f. Often talks excessively.
g. Often blurts out an answer before a question has been completed (e.g., completes people's sentences; cannot wait for turn in conversation).
h. Often has difficulty waiting his or her turn (e.g., while waiting in line).
i. Often interrupts or intrudes on others (e.g., butts into conversations, games, or activities; may start using other people's things without asking or receiving permission; for adolescents and adults, may intrude into or take over what others are doing).

Figure 1. Inattentive/Hyperactive Symptoms of ADHD

Does ADHD Persist into Adulthood?

For most of the last century, the literature focused on ADHD as a disorder of childhood. However, researchers and clinicians have come to recognize in increasing numbers that ADHD persists into adulthood in some instances (Bachorowski, & Newman, 1985; Remschmidt, 2005; Montes, Garcia, & Ricardo-Garcell, 2007; Rabiner, Anastopoulos, Costello, Hoyle, & Swartzwelder, 2008).

Faraone and Biederman (2005) estimated that the prevalence of adult ADHD is 2.9% with 16.4% of adults meeting partial criteria. Ingram, Hechtman, and Morgenstern (1999) found that ADHD often follows a lifelong course, with as many as 60% of persons diagnosed with ADHD in childhood continuing to exhibit symptoms in adulthood, although only half of those continue to meet full diagnostic criteria. While the core symptoms of hyperactivity tend to decrease with age, problems related to inattention persist, and secondary problems, such as social skills deficits, may actually increase in severity (Sprafkin, Gadow, Weiss, Schneider, & Nolan, 2007). Murphy and Barkley (1996) found that adults with ADHD continue to experience myriad problems related to everyday living, including depression, anxiety, substance use disorders, motor vehicle tickets and accidents, and employment instability. Adults with ADHD were more likely to drop out of college, get suspended from school, get divorced, and have difficulty establishing and maintaining close personal relationships.

In addition, many adults who were not diagnosed as children present for evaluation upon entering the workforce/college or during other transitional periods where they are required to exercise greater levels of self-discipline and internal motivation than in primary and secondary education settings where their parents and teachers provide external sources of motivation

(Ramsey & Rostain, 2008). Individuals with ADHD who were not identified in childhood exhibit similar, sometimes even more severe, patterns of secondary deficits and psychiatric symptoms compared to those who were identified and treated (Ingram, et al, 1999). Furthermore, since the diagnostic criteria for ADHD were developed for children, correctly diagnosing adults without a history of childhood diagnosis can be problematic. DSM-5 has increased the age before which symptoms must be present from 7 to 12 years of age to make retrospective self-report easier (APA, 2013), and several measures examining retrospective symptoms of ADHD in childhood (Hill, Pella, Singh, Jones, & Gouvier, 2009; Glockner-Rist, Pedersen, & Rist, 2013) or current symptoms appropriate to adults (Belendiuk, Clarke, Chronis, & Raggi, 2007; Rodriguez & Simon-Dack, 2013) have been developed. Several studies have demonstrated that adults are reliable and valid self-reporters of ADHD symptoms on questionnaires, rating scales, and diagnostic interviews compared to collateral sources of information (Belendiuk, et al., 2007), although self-report does not always reflect functional impairment (Burlison & Dwyer, 2013).

Clinical Assessment of ADHD: What Is The Role of Neuropsychology?

Attention-Deficit/Hyperactivity Disorder is diagnosed primarily on the presence or absence of a specific number of symptoms of either inattention, hyperactivity/impulsivity, or both. These symptoms are routinely assessed through parent, teacher, or self-report questionnaires, and symptoms must be present in more than one major setting. The American Academy of Pediatrics also recommends an assessment for comorbid or alternative diagnoses, and behavior therapy as part of the first line treatment for ADHD (AAP, 2011), although these guidelines were followed by less than half of physicians and psychologists who responded to a

survey about their diagnostic practices (Chan, Hopkins, Perrin, Herrerias, & Homer, 2005). Morley (2010) reports that ADHD diagnosis varies along socio-economic status with higher rates of ADHD diagnosed among children from lower SES backgrounds. However, ADHD diagnostic precision is less consistent at the lower range of the SES spectrum, as more children with other behavior problems are mislabeled with ADHD, while many children with qualifying symptoms are never presented for evaluation. Froelich and colleagues (2007) observed that only 47.9% of children who met criteria for ADHD had been diagnosed, while only 32.0% received treatment. Both Morley (2010) and Froelich, et al. (2007) concluded that this diagnostic variability may be due to physician unfamiliarity, and both found that using standardized symptom checklists or formal behavior observations improved diagnostic accuracy.

While neuropsychological testing is not required to make a diagnosis of ADHD, there are several ways that neuropsychological assessment can potentially enrich our understanding of ADHD. Neuropsychological assessment can help identify possible ADHD profiles or syndrome clusters in order to facilitate differential diagnosis of similar or commonly comorbid disorders (e.g., anxiety, bipolar disorder, neurocognitive disorder), and can help quantitatively evaluate the relative effectiveness of different interventions/accommodations or long-term prognosis. Neuropsychological assessment has also been used to identify and investigate the core neurocognitive dysfunctions believed responsible for the symptoms of ADHD.

Identifying syndrome clusters or profiles.

According to Rutter (1978), a syndrome is only useful if it predicts something beyond a description of the symptoms. There have been many attempts to identify a neuropsychological profile of ADHD to enhance diagnostic specificity or facilitate rehabilitation plans (Snow &

Sapp, 2000; Assesmany, McIntosh, Phelps, & Rizza, 2001; Mayes & Calhoun, 2004, 2006; Thaler, Bello, & Etcoff, 2012). Profile analysis of Wechsler subtests have been proposed, but replication has proven problematic (Watkins, 2003; Ek, et al, 2007). One major drawback of profile analysis is the relatively lower reliability of Wechsler subtests compared to the more stable composite scores. This difference in reliability negatively impacts the validity of subtest profiles (Watkins, Glutting, & Youngstrom, 2005; Strauss, Sherman, & Spreen, 2006). Another drawback to subtest profile analysis is the assumption that subtests were chosen based on a pathognomonic relationship to a particular cognitive function. While that may be true for some measures, such as the Woodcock-Johnson III (McGrew & Woodcock, 2001), in the case of the Wechsler scales, subtests were chosen based on the strength of relationship to overall intelligence and not for individual discriminability (Wechsler, 1997, 2008). According to Brody (1985), subtest analysis is only beneficial if the improvement in predictive validity over the composite score alone is sufficient to accept the loss in reliability of that prediction. With the Wechsler scales, the index scores are both more reliable and valid than the subtest scores (McDermott, Fantuzzo, & Glutting, 1990). Despite the flaws in profile analysis demonstrated in the research literature, the approach is still favorably viewed by practitioners, who find the technique useful for differential diagnosis, but not for rehabilitation planning (Pfeiffer, Reddy, Kletzel, Schmelzler, & Boyer, 2000).

The use of continuous performance tasks (CPT) to identify weaknesses associated with ADHD developed in the 1990s and continues today (Losier, McGrath, & Klein, 1996). A continuous performance task is a computer-administered assessment in which stimuli are presented rapidly and sequentially, and the examinee must maintain vigilance to respond

appropriately for the duration of the test, typically 10-15 minutes. CPTs measure vigilance and reaction time, as well as inhibition and impulsivity when the tasks are made more complicated (e.g., variable stimulus presentation intervals may catch a respondent who is rhythmically pressing the response key, while distinguishing between target and distractor stimuli requires examinees to respond with caution). Advokat, Martino, Hill, & Gouvier (2007) demonstrated that Connor's CPT (Connors, 2004) reliably differentiates between ADHD and normal controls or individuals with other psychiatric conditions. However, the CPT did not discriminate between individuals with ADHD and those with other learning disabilities. Riccio, Reynolds, and Lowe (2001) say that CPTs provide evidence of impairment consistent with ADHD, but lack specificity and sensitivity, and are not the best tool for diagnosing ADHD.

Differential Diagnosis of ADHD.

Psychological testing is a useful tool to aid in differential diagnosis of ADHD from other neurodevelopmental or neurocognitive disorders. Disorders with symptom clusters that overlap significantly with ADHD include specific learning disorders, mood and affective disorders (such as major depressive disorder or generalized anxiety disorder), and mild neurocognitive disorder. Any assessment for ADHD should rule-out the presence of these conditions. Therefore, a discussion of the presenting symptoms for these conditions and a brief survey of how these disorders are distinguished from ADHD are relevant to the present investigation.

Differential diagnosis of ADHD vs. Learning Disorders. Individuals with learning disorders constitute the largest segment of the school-aged population who qualified as disabled, and comprise 41% of those receiving services through the Individuals with Disabilities Education Act of 2004 (IDEA 2004; IDEA Part B Child Count, 2011). Learning disorders (LD)

can manifest as a significant difficulty in academic domains such as reading, writing, listening, speaking, or mathematical abilities (Osmon, Patrick, & Andresen, 2008). Learning disorders often co-occur with ADHD, but can be differentiated from ADHD by the presence of academic domain-specific deficits.

The core dysfunction in learning disorders is heterogeneous—even within categories. For example, functional academic impairment observed in Specific Learning Disorder, With impairment in reading, may include deficits in reading comprehension, word reading accuracy, or reading fluency (e.g., reading speed), each involving multiple centers of cognition reflected in the different strategies for teaching reading. Bottom-up processes, such as phonemic awareness and sound blending, are utilized in phonics reading programs which emphasize assembling words from component syllables. Top-down processes, such as word recognition and semantic context, form the basis of the whole-language instructional philosophy which relies on students recognizing combinations of letters as semantically meaningful pictures. Bottom-up phonetic reading is especially important in the early stages of learning to read, when reading is not automatic and the child recognizes few words. However, as reading becomes more automatic, fluency increases using top-down, sight reading methods. This top-down fluency is demonstrated by the common illusion that scrambling the letters within a word does not affect reading comprehension in fluent readers, especially where words retain their familiar shape.

Although the core dysfunction in learning disorders may be heterogeneous, a learning disorder can be distinguished from ADHD by the presence of significant impairments primarily within one domain of academic functioning. Perhaps the most widely used method for identifying learning disorder is the aptitude-achievement discrepancy analysis. This technique

involves comparing an individual's score on an aptitude test, such as the Wechsler Intelligence Scales for Children (WISC), with their score on an achievement test, such as the Wechsler Individual Achievement Test (WIAT; Reynolds & Livingston, 2012). If the individual's achievement in a specific domain (e.g., reading, math, etc.) is significantly lower than their measured intelligence, then this represents impairment in that academic domain and satisfies criterion B for Specific Learning Disorder in the *Diagnostic and Statistical Manual* (5th ed.; *DSM-5*; APA, 2013).

However, there are problems with the use of aptitude-achievement discrepancies to diagnose learning disorders. Vukovic and Siegal (2006) summarized these difficulties best when they concluded that IQ-achievement discrepancy: a) over-identifies children with high IQ but low achievement, whose deficits may better be accounted for by environmental disadvantage or motivational issues; b) under-identifies children w/low IQ who present with clear processing problems associated with LD, but whose low aptitude make discrepancies narrower; and c) leads to the practice of "waiting to fail" in which children are retested at intervals until their aptitude-achievement discrepancy reaches a level of clinical significance, by which time they have fallen substantially behind their peers in the deficient domain. Because of these limitations, the clause, "despite the provision of interventions that target those difficulties, p. 66" was added to criterion A and criterion C "The learning difficulties begin during school-age years but may not become fully manifest until the demands for those affected academic skills exceed the individual's limited capacities, p. 67" were added to Specific Learning Disorder in the DSM-5 (APA, 2013).

These additions to the criteria for Specific Learning Disorder reflect changes to the literature brought about by a second approach to identifying a child with LD: response to

intervention (RTI). RTI involves 3 tiers of interventions and progress monitoring through curriculum-based assessment, or measuring children's achievement through the material they are learning in school, rather than individually-administered standardized tests of achievement. At the first tier, each student receives research-supported general classroom instruction. This assures that students who fall behind on curriculum-based measures are not falling behind due to lack of appropriate instructional opportunities. The students in Tier 1 whose performance on curriculum-based measures indicates deficits (10-20% of students, estimated) in achievement are given additional supports, such as peer-tutoring opportunities or individually-tailored instruction for 30 minutes per day. Progress is monitored through curriculum-based assessments, and those who do not respond to Tier 2 services (5-10% of students, estimated) receive Tier 3 services, which include intensive interventions and a special education referral to develop an Individualized Education Plan (IEP). The rationale is that students who do not respond to individualized instruction have a learning disorder, regardless of aptitude-achievement discrepancy (Kavale, Fuchs, & Scruggs, 1994; Gresham, 2007).

Proponents of RTI have argued that RTI identifies children with LD at an earlier age, before they have fallen behind their peers, and that the tier system benefits at-risk students as well as those with an LD (Fuchs, & Fuchs, 2006; Kavale, Kauffman, Bachmeier, & LeFever, 2008). Criticism of RTI centers on the additional training and resources necessary to implement RTI in the schools, the delay in identifying students needing special education, and difficulties reliably discriminating between LD and other developmental causes of low achievement (McKenzie, 2009; Reynolds, & Shaywitz, 2009; Wertz, Lambert, & Carpenter, 2009).

However, criticism associated with RTI's poor discriminability as a stand-alone

technique, while serious, is not surprising. It has never been good practice to base clinical decisions on any single piece of evidence. When incorporated into a clinical service delivery system, however, RTI offers a set of beneficial assessment and intervention approaches particularly applicable in early education. By contrast, while RTI may provide greater sensitivity and informs intervention with school-aged children, the criticisms against the aptitude-achievement discrepancy technique, such as "waiting to fail," do not apply to the assessment of adults with LD who were not identified as children (Reynolds, & Livingston, 2012; Osmon, et al., 2008), and the IQ-achievement discrepancy technique possesses greater sensitivity and more diagnostic specificity, particularly for adult populations.

Differential diagnosis of ADHD vs. Mood and Affective Disorders. ADHD shares symptoms with several other disorders which must also be considered in order to make a proper rule-out. However, unlike ADHD, which is a persistent, neurodevelopmental disorder, many of the symptoms of inattention, hyperactivity, or impulsivity which are observed along with disorders are cyclical (e.g., major depression, mania), occur within the context of the psychiatric disorder or along with significant symptoms related to that condition (e.g., dysthymia, anxiety, psychosis), or possess a discrete observable etiology (e.g., substance use, medication-induced symptoms of ADHD, or neurocognitive disorders). Each of these conditions can be ruled out or examined with background information, a diagnostic clinical interview, and supplemental testing.

Individuals with depression may present with restlessness and difficulty concentrating, but in a depressive disorder, these symptoms occur along with sadness or irritability, guilty feelings, and anhedonia, and wax and wane with those symptoms. By contrast, restlessness and

difficulty concentrating observed in ADHD are persistent.

Individuals with a bipolar disorder may experience impulsivity, hyperactivity, and distractibility. However, the presence of these symptoms is cyclical, and occurs within the context of a mood episode, diminishing between episodes. Furthermore, symptoms of bipolar disorder are rare in preadolescents, whereas symptoms of ADHD must be present before age 12, and should be persistent—not cyclical.

Anxiety disorders share symptoms of inattention and restlessness with ADHD. However, inattention in anxiety often takes the form of anxious rumination and worry, rather than distractibility and interest in novel or stimulating activities.

Differential diagnosis of ADHD vs. Other Conditions. Symptoms consistent with ADHD are sometimes observed during psychotic episodes. However, ADHD is a persistent condition, and should not be diagnosed if the symptoms occur cyclically, or exclusively during the course of a psychotic episode. Similarly, while many individuals with ADHD—particularly those who were not diagnosed in childhood, attempt to self-medicate using alcohol or illicit substances, ADHD is not diagnosed in instances when symptoms of inattention, hyperactivity, or impulsivity occur exclusively within the context of substance intoxication or withdrawal.

Finally, individuals with Mild Neurocognitive Disorder may present with a clinical syndrome similar to that of ADHD. Mild Neurocognitive Disorder can be differentiated from ADHD by the presence of known brain injury or laboratory findings consistent with mild cognitive impairment, a precursor to dementia, as well as an absence of premorbid deficits or the absence of symptoms associated with ADHD before age 12 years.

Extraneous variables and covariate analysis.

Several confounding variables have been implicated in previous studies of ADHD with mixed or null outcomes. For example, similar disorders that occur comorbidly with ADHD, such as depression, anxiety, and specific learning disorder were theorized to independently impact dependent measures of executive dysfunction. Demographic variables including age, sex, race, and SES have also been implicated. One approach to extraneous confounding variables is to statistically control for them using covariate analysis.

An extraneous variable is appropriate for inclusion as a covariate when it is correlated with the dependent variables, but independent of the predictor variables. A variable that correlates strongly with the independent variable, but does not correlate with the dependent variable, will not have a substantial impact on the results of a significance test. For example, if depression and ADHD were highly correlated, but depression was negligibly correlated with response time, then response time is an effective way of discriminating between the two conditions, and no statistical control is needed. If, on the other hand, an extraneous variable correlates strongly with the dependent variable, but not with the independent variable, that variable introduces a source of measurement error if not controlled. For example, if ADHD and cognitive ability were negligibly correlated, but cognitive ability was highly correlated with number of errors made on a test, then a significant amount of the difference in errors would be due to cognitive ability instead of ADHD, and, while removing the shared variance between cognitive ability and ADHD decimates the remaining variance due to ADHD, it removes a tremendous amount of confounding error, and controlling for it statistically permits the examiner to detect the actual effect of ADHD. Finally, if a variable correlated strongly with both the

independent variable and the dependent variable, then, while eliminating it reduces a lot of shared variance, doing so eliminates a significant amount of error variance. For example, if ADHD and reading fluency were highly correlated, but reading fluency was also correlated highly with response time, removing the shared variance between reading fluency and ADHD would increase the statistical power of the comparison between ADHD and response time by removing the variance due to reading fluency.

Identifying the Core Dysfunction in ADHD

Current theory assumes that, while ADHD is heterogeneous in symptom presentation, it is likely to be homogeneous (or perhaps dichotomous) in core neuropsychological dysfunction. Neuropsychological tests have been used to cultivate and examine several competing theories of core ADHD dysfunction. Two of the foremost theories are the executive dysfunction theory, and the state regulation theory (Johnson, Wiersema, & Kuntsi, 2009).

Executive Dysfunction Theory of ADHD.

The theory that ADHD is a result of executive dysfunction can be traced back to Barkley (1997) and has found widespread support in the literature (Chhabildas, Pennington, & Willcutt, 2001; Pasini, Paloscia, Alessandrelli, Porfirio, & Curatolo, 2007; Brown, 2008; Boonstra, Oosterlaan, Sergeant, Kooij, & Buitelaar, 2010). Specific deficits of executive functions reported in ADHD have included working memory, processing speed, self-regulation of affect-motivation-arousal, internalization of speech, and reconstitution (behavioral analysis and synthesis), and particularly set shifting and response inhibition (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Alvarez & Emory, 2006). These deficits center on the dopaminergic and noradrenergic neurons of the anterior cingulate gyrus in the prefrontal cortex (Arnsten, Steere, &

Hunt, 1996; Bush, et al, 1999).

Despite the theory's widespread acceptance, meta-analyses have been inconclusive (Sergeant, Geurts, & Oosterlaan, 2002; Hervey, Epstein, & Curry, 2004; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Sergeant and colleagues (2002) found that, while executive dysfunction is common in individuals with ADHD, the pattern is not consistent between studies or specific to ADHD. Similarly, Willcutt and colleagues (2005) concluded that executive dysfunction is neither necessary nor sufficient to cause all of the cases of ADHD, but is instead one component of a complex neuropsychological picture.

Other researchers have concluded that the problem lies not with the theory, but with the ways executive functions are measured in the literature. Brown (2006) stated that the executive functions are complex, emergent processes that are not reducible to neuropsychological tests of executive functioning, and that the focus should be on practical skills and complex tasks. Following that recommendation, Torralva, Gleichgerrcht, Lischinsky, Roca, and Manes (2013) administered traditional executive tasks along with more difficult and ecologically valid executive tasks and found that only the non-traditional tasks were able to discriminate between high functioning adults with ADHD and normal controls. They concluded that for many high functioning adults with ADHD, standard neuropsychological testing may not be sensitive enough to detect executive dysfunction. However, by concluding that executive dysfunction is ADHD is unobservable by standard neuropsychological tests, the authors may have inadvertently weakened the theory. Since it was evidence of impairment on those tests which led to the executive dysfunction theory in the first place, eliminating the need for measurable deficits in executive functioning also removes the theory's falsifiability, rendering it scientifically tenuous.

State Regulation Theory of ADHD.

The State Regulation theory of ADHD was developed by Sergeant (2000) out of the Cognitive-Energetic model of Sanders (1983). According to this theory, there are three levels of cognitive processes affected by ADHD: a) lower cognitive processes such as encoding, central processing and response organization distributed through the cerebellum and brainstem; b) mid-tier processes such as arousal, activation, and effort centered around the ventral tegmental area of the midbrain; and c) higher order cortical executive functions centered on the anterior cingulate gyrus. Sergeant, in investigations of the Cognitive-Energetic model, has identified the principle deficits associated with each of these levels as deficits in motor organization (lower-tier), deficits in arousal and activation (mid-tier), and deficits in response inhibition (higher-order; Sergeant, 2000; 2005).

The State Regulation theory of ADHD proposes that inefficiency at each of the three levels of cognitive processes, but particularly at mid-tier arousal and activation, contributes to the pattern of deficits observed in ADHD. According to this model, arousal is defined as a systematic physiological response to input, while activation refers to a long-lasting voluntary readiness for action. Accordingly, the deficits observed in ADHD are due to a failure to be adequately aroused by, and subsequently engage with, easy or boring tasks. It is argued that individuals may become hyperactive as a form of self-stimulation. In contrast, when the task is stimulating or rewarding, a threshold of arousal is crossed, and the individual is able to engage appropriately with the task (Kuntsi, McLoughlin, Asherson, 2006). This may explain why individuals with ADHD are able to hold their attention for extended periods of time for highly stimulating activities such as video games or watching television, but not while working on

school assignments.

In support of this model, individuals with ADHD have been found to respond with more variability and were found to have a rapid decline in efficiency during tasks with slower stimulus presentation times, but responded more consistently and accurately during tasks with faster presentation times (Scheres, Oosterlaan, & Sergeant, 2001; Van der Meere, 2002). This may explain why researchers investigating attention deficits in adults with ADHD found meaningful differences between ADHD groups and controls for sustained attention tasks, but not consistently for focused attention (Marchetta, Hurks, De Sonneville, Krabbendam, & Jolles, 2008).

Deficits in individuals with ADHD predicted by the state regulation theory have been linked to fMRI imaging which showed differential activation on both forward and backward digit span tasks, indicating that even when there are no systematic differences in psychological test performance, for example, on the simpler digits forward task, there may be systematic differences in the way the ADHD brain responds during both simple and complex cognitive operations (Hale, Bookheimer, McGough, Phillips, & McCracken, 2007). Poor state regulation has also been implicated as contributing to ADHD symptoms in children under age 7 years (Berwid, et al., 2005).

How these two theories compare and contrast.

While both theories hypothesize neurocognitive deficits associated with the anterior cingulate gyrus, the two differ in their predictions, the outcomes of which have significant physiological implications. The executive dysfunction theory of ADHD argues that the symptoms of inattention and hyperactivity in ADHD are primarily due to general deficits in

executive functions associated with the dorsal anterior cingulate gyrus (Allman, et al, 2001), especially set shifting and response inhibition. Consequently, one would expect an individual with ADHD to perform consistently poorer on tasks relying heavily on executive control, relative to more straightforward attentional tasks. Conversely, the state regulation theory of ADHD argues that the symptoms of ADHD are primarily associated with deficits in motivation and primary arousal, associated with the ventral anterior cingulate gyrus (Allman, Hakeem, Erwin, Nimchinsky, & Hof, 2001), resulting in inadequate arousal and activation of attentional systems in response to less stimulating or challenging stimuli. Consequently, researchers have looked for poorer performance on slower, monotonous tasks than on tasks which demand more varied and frequent responding.

CHAPTER 2

PURPOSE OF STUDY

The research so far has provided mixed evidence for supporting the outcomes predicted by both the executive dysfunction and the state regulation theory. However, there is a dearth of studies directly contrasting predictions of state regulation theory against those made by the executive dysfunction theory. This study tackles that challenge by using a cognitive task specifically developed to produce readily interpretable outcomes as predicted only by one or the other of the two theories, but not both. Both theories describe deficit profiles occurring along the pathway between the anterior cingulate gyrus in the prefrontal cortex and the ventral tegmental area in the midbrain (Bush, et al, 1999; Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Tomasi & Volkow, 2014) limiting opportunity to demonstrate a true double dissociation (Garcia & Koelling, 1966), nonetheless the two theories differ enough in their emphasis of deficits associated with one region over the other, for the sake of hypothesis testing within the present research, a double dissociation paradigm was proposed.

While initial studies for the executive dysfunction theory of ADHD offered promising support for this theory, subsequent meta-analyses conducted using the past two decades of research have been less conclusive. One likely culprit may be the potentially heterogeneous nature of ADHD. Consequently, there has been a recent shift away from DSM categorical taxonomy toward a symptom-level analysis with the expectation that symptom-level examination will reveal more consistent syndromes within or across current diagnostic categories. Therefore, the present study conducted symptom-level analyses based on published NIMH RDoC paradigms to determine whether specific symptoms or combinations of symptoms align with the

predicted results from one or another of the theories under investigation.

Hypotheses

In global terms, this study seeks to examine which theory of ADHD, Executive Dysfunction or State Regulation, more consistently characterizes individuals with symptoms consistent with ADHD, while also examining the potential influence of confounding demographic variables, symptoms of mood and affective disorder, and individual differences in cognitive functioning.

If correct, the State Regulation theory offers the basis for hypothesizing a pattern of deficits in which as degree of ADHD increases, individuals will display relatively poorer performance (i.e., longer reaction times and more errors) for the less arousing tasks (i.e., longer inter-stimulus interval, normal stimuli) relative to the more difficult tasks (i.e., shorter inter-stimulus interval, novel stimuli). Conversely, the executive dysfunction theory would be supported if degree of ADHD was significantly related to poorer performance (i.e., longer reaction times and more errors) on the high inhibition trial of the CPT.

Ory (2015) reported results suggestive of a possibly reliable relationship among diagnostic categories and psychoeducational profiles, based on analysis of an ongoing database of evaluations held securely at the LSU Psychological Services Center (Table 1). The conduction and findings of that previous study helped to refine the methodology used here, and led to the construction of a reaction time test specifically designed to address the experimental hypotheses. The findings of Ory (2015) were likely constrained by many methodological limitations that were addressed in the present research. Ory used an archive of data collected over 20 years, and diagnostic categorizations were made by dozens of clinicians. In the present study, diagnostic

Table 1. Mean and SD of WAIS-III Digit Span and Trails by diagnostic group from an archival database.

Measure	No Diagnosis *n* = 43	ADHD, NOS *n* = 53	ADHD, Inattentive *n* = 105	ADHD, Hyperactive/Combined *n* = 62
Digits Forward total raw score	11.02 (1.99)	10.87 (2.47)	10.17 (2.33)	9.87 (2.04)$_w$
Trails A t-scores	43.35 (7.55)	43.28 (9.58)	42.40 (11.64)	42.06 (11.87)
Digits Backward total raw score	7.14 (2.02)	7.62 (2.83)$_s$	6.82 (2.35)$_w$	6.50 (2.01)$_w$
Trails B t-scores	45.93 (10.18)	46.32 (10.07)$_s$	44.90 (10.14)	40.86 (11.32)$_w$

s = strength relative to No Diagnosis group (these were not significant)
w = weakness relative to No Diagnosis group (means significantly different at .05 level)
(Table reproduced from Ory, 2015)

criteria were assessed explicitly, and diagnostic category was assigned uniformly based on current DSM-5 criteria. Additionally, the subjects included in that earlier study were pulled from a clinic database; even the no diagnosis "controls" had presented at the clinic for assessment of perceived cognitive or emotional dysfunction. Participants in the present study were selected from a general university undergraduate pool, and are a more broadly representative control group and permit more generalizable findings. Finally, since the 2015 study used a clinical archive, analyses were limited to measures that were given to a wide number of clients, in this case, WAIS-III Digit Span and the Trail Making Test Parts A and B. The present research afforded the opportunity to develop a task using paradigms that better accessed the principle deficits attributed to ADHD according to each theory.

Due to the inconsistent state of the literature, and as suggested in the "future directions" from the Ory (2015) preliminary findings, this study adopted the premise that ADHD is a neurocognitively heterogeneous category of functionally similar disorders (Wahlstedt, Thorell, & Bohlin, 2009; Forslund, Brocki, Bohlin, Granqvist, & Eninger, 2016). Therefore, specific

symptoms of ADHD, or symptoms of other disorders sharing overlap with ADHD, may predict different patterns of deficits on tasks sensitive to executive dysfunction or state regulation problems, as compared to the deficit profiles revealed when ADHD is analyzed by syndrome (or diagnosis sub-type). To state the premise explicitly, different theories may be correct for different subtypes of ADHD. As such, one, the other, both, or neither theory of ADHD may be supported within the present sample. Follow-up investigation using the present data as a guide will be necessary to test emergent hypotheses about subsyndromes.

The hypotheses are:

H_{0a}: If the patterns predicted by the Executive Dysfunction or State Regulation theories do not occur among the dependent variables, neither theory earns support. The Null here is tested by looking for more errors or slower response times as a function of ADHD for high inhibition trials (executive dysfunction) vs chance; and also looking for more errors or slower response times as a function of ADHD for low arousal trials (state regulation) vs. chance.

H_{0b}: If both the Executive Dysfunction and State Regulation predicted patterns among the dependent variables occur, both theories receive some support. The Null test here is the same as used above

H_1: If more errors or slower response times are observed as a function of ADHD for high inhibition trials, but not for low arousal trials, the Executive Dysfunction theory is supported over the State Regulation theory. The Null test here is the same as used above.

H_2: If more errors or slower response times are observed as a function of ADHD for low arousal trials, but not for high inhibition trials, the State Regulation theory is supported over the Executive Dysfunction theory. The Null test here is the same as used above.

Each of these will be tested using data examining 1) symptom frequency, 2) symptom severity, and 3) diagnostic criteria.

To test these hypotheses, three sets of mixed factorial analyses of covariance (ANCOVA) were planned. However, analysis of covariance was not used due to weak correlations between covariates and dependent variables. A continuous performance task was programmed to manipulate inhibition and arousal within subjects using RDoC paradigms (i.e., stimulus-response compatibility and conflicting and contralateral motor response paradigms were manipulated together as one independent variable for inhibition; interstimulus interval and stimulus novelty was manipulated independently as two separate independent variables of arousal). Response time and number of errors served as dependent variables. ADHD symptom data were collected, and used to construct between subjects measures of ADHD in three sets of analyses: Analysis 1 defined degree of ADHD as symptom frequency (i.e., number of symptoms of either inattention or hyperactivity endorsed as occurring often/frequently); Analysis 2 defined degree of ADHD as severity of ADHD symptoms (i.e., sum of severity Likert ratings within inattentive and hyperactive domains); Analysis 3 defined degree of ADHD according to DSM-5 criteria for ADHD (e.g., Five or more symptoms of either inattention or hyperactivity endorsed as occurring often/frequently at a severity of 3-4 out of 4, with an onset in childhood satisfied criteria for ADHD-I or ADHD-H respectively, while 4 symptoms endorsed at a severity of 4 out of 4 was categorized as "possible ADHD"). The examiner expected more errors and longer response times for individuals with greater degrees of ADHD hyperactivity on high inhibition trials, and more errors and longer response times for individuals with greater degrees of ADHD inattention on low arousal trials.

CHAPTER 3

METHOD

Participants

Participants were recruited from a large state university and received course credit for their participation. In total, 200 participants were recruited, 9 participants voluntarily withdrew without completing the entire protocol, and 25 were discarded unilaterally from all analyses because they abandoned the continuous performance task before it was completed, as indicated by their failing to respond to at least 80% of presentations on the CPT. A further six participants were excluded from the final analyses as outliers because their performance during the high inhibition trial indicated that they had not read or understood the instructions. Of the remaining 160 participants, there were 26 males and 134 females. The mean age of the sample was 20.43 years (3.07 SD, range = 18-52). The sample included 124 white/Caucasian students, 23 Black/African-American students, and 1 Asian student. Six participants identified as Hispanic/Latino, 4 identified as multi-ethnic, and 2 declined to answer. The sample is broken down by race and sex in Table 2.

Power analysis was conducted to determine the minimal sample size necessary to achieve a power of .80. A conservatively estimated small effect size was used based on effect sizes from Ory (2015) and a review of previous studies that examined the differences among subgroups of ADHD subjects using continuous performance errors and response time (Wilcutt, et al, 2005; Sergeant, et al, 2002; Geurts, et al, 2005). A total required sample size of 180 was computed using G*Power 3.1.9.2 testing for ANCOVA: Fixed effects, main effects and interactions (Faul, Erdfelder, Lang, & Buchner, 2007).

Table 2. Sample demographics displayed by sex and cultural identity.

	Male	**Female**	**Row Total**
Asian/Pacific Islander	0	1	1
Black/African-American	4	19	23
Hispanic/Latino/American Indian	1	5	6
White/Caucasian	20	104	124
Multi-Ethnic	0	4	4
Declined to Answer	1	1	2
Column Total	26	134	160

Procedure

The experiment was conducted 100% online. Despite the potential for unaccountable interruptions with resulting increases in sampling error (Reips, 2002), research examining differences in the reliability and validity of continuous performance tasks administered over the internet has indicated acceptable construct validity and test-retest reliability between home- and laboratory-administered tests (Raz, Bar-Haim, Sadeh, & Dan, 2014). Some authors assert that testing in an uncontrolled setting actually offers increased ecological validity, as naturally occurring environmental distractions would be expected to impact individuals disparately according to their degree of ADHD impairment (Torralva, et al, 2013).

Upon enrolling in the experiment online, participants were provided with a link and access code to connect to the experiment's secure page in LSU's Community Moodle. Participants were given opportunity to review the consent form (Appendix A) online. Once they provided consent, the rest of the experiment became available. Participants then completed a background and symptom questionnaire accessed through Qualtrics, a 9-item short form (Bilker,

et al., 2012) of the Raven's Standard Progressive Matrices (RSPM) to screen for general cognitive functioning, and an online reading fluency task adapted from the Woodcock-Johnson-III Achievement Battery (McGrew & Woodcock, 2001) to screen for symptoms of Reading Disorder. These tasks were administered in order to permit statistical control for general cognitive functioning and reading speed, if necessary. The experimental task was a continuous performance test programmed for this experiment in JavaScript using the JSPsych library of plug-ins (de Leeuw, 2015). Once all tasks were completed, participants were assigned additional course credit for their participation. Moodle is a secure learning management system compliant with Family Educational Rights and Privacy Act of 1974 (FERPA) security standards. To further ensure security of data and the anonymity of participants, each participant was given a subject number along with the link and access code to the Moodle site. All parts of the experiment accessed outside of the secure Moodle site were accessed using that subject number only, rather than any identifying information. Participants' names were attached to their subject numbers through the consent form in Moodle, but were not included together externally.

Materials

Background and Symptom Questionnaire. The background and symptom questionnaire (Appendix B) was administered using Qualtrics and included demographic and symptom questions. The questionnaire was developed specifically for this experiment, and used explicit DSM-5 criteria for ADHD, Major Depressive Episode, and included symptoms of panic attack, social anxiety, and generalized anxiety disorder. For each symptom, the questionnaire asked about frequency (e.g., rarely, sometimes, often), and severity (1-4 Likert scale). Additionally, for ADHD, participants were asked about symptom onset (i.e., within the past 6 months, past

several years, since childhood/whole life). For depression, participants were asked whether they were experiencing the symptoms currently or if they experienced them in the past, and, if currently experiencing the symptom, for how long the symptom had been present (i.e., few days, few weeks, and several years). For the symptoms of anxiety, participants were asked about specific triggers and were provided with an open response format to list specific triggers. They were also asked whether the anxiety was acute (e.g., passing within minutes) or continuous (e.g., present more often than not each day). In addition, information on demographic variables (i.e., age, sex, cultural identity, years of education, occupation, and whether their hometown was a rural, suburban, or urban community) presumed to have confounded results of other ADHD studies was collected in order to statistically control for their influence, if necessary.

Experimental Continuous Performance Test. Disinhibition and arousal were assessed within subjects using a continuous performance task (CPT). The CPT was chosen due to its frequent use in the ADHD literature as a technique for identifying deficits related to ADHD (Losier, et al, 1996; Epstein, et al, 2003; Berwid, et al, 2005; Oberlin, Alford, & Marrocco, 2005; Advokat, et al, 2007; Belendiuk, et al, 2007; Marchetta, et al, 2008; Albrecht, Uebel-von Sandersleben, Wiedmann, & Rothenberger, 2015) and because the nature of the task permitted measurement of several key dependent variables of interest and the ability to effect manipulation of multiple independent variables within a single 10-minute administration.

Inhibition manipulation. Inhibition was measured using two paradigms identified by RDoC as well supported: stimulus-response compatibility and conflicting and contralateral motor response. These two paradigms were manipulated concurrently to enhance sensitivity through redundancy, and for the purposes of this study are inseparably one inhibition manipulation.

Stimulus-response (S-R) compatibility is the degree to which a presented stimulus is compatible with the required response (Simon & Rudell, 1967). Research has shown that response times are slower when a stimulus is presented to the opposite visual field from the required motor response than when the stimulus and response are on compatible sides (Aron, 2007). According to the executive dysfunction theory, individuals with ADHD have a primary deficit in inhibition, and are expected to have more difficulty than individuals without an attentional disorder on stimulus incompatible trials (Mostofsky, Newschaffer, & Denckla, 2003; Mahone, Powell, Loftis, & Goldberg, 2006; Wodka, et al., 2007). For this experiment, during the low inhibition trial, participants responded to stimuli presented on the left side of the screen by pressing the ← key and stimuli presented to the right side of the screen by pressing the → key. For the high inhibition trial, participants responded to stimuli presented on the left side of the screen by pressing the → key and stimuli presented to the right side of the screen by pressing the ← key. By administering both blocks to each participant rather than between subjects, we introduced a conflicting and contralateral motor response task, another inhibition paradigm, which has demonstrated slower response times and more errors when the necessary response conflicts with expectations and experience (DeSoto, Fabiani, Geary, & Gratton, 2001). After first practicing responding to the congruent side, participants should find it difficult to respond to the contralateral side during the second trial. Individuals with ADHD should find this particularly more difficult, according to the executive dysfunction theory.

First arousal manipulation—Interstimulus Interval. The RDoC paradigm for arousal is psychomotor vigilance, which has been used in studies evaluating the state regulation theory, typically by using slower stimulus presentation times to manipulate arousal (Van der Meere &

Sergeant, 1988a & 1988b; Van der Meere, Vreeling, & Sergeant, 1992; Scheres, et al, 2001). For the CPT program used in the present experiment, interstimulus intervals (ISI) of 1000ms, 1500ms, and 2500ms were chosen. For the analyses discussed below, 1000ms and 2500ms ISI were used to represent high and low arousal blocks respectively in order to increase sensitivity and power by focusing on extreme differences. For purposes of this report, the data from the 1500 ms trials were not included in the analyses.

Second arousal manipulation—stimulus novelty. The second manipulation of arousal used in the present experiment is the oddball paradigm. The oddball paradigm involves the presentation of a rare stimulus after a series of standard stimuli which has been demonstrated to produce a momentary spike in arousal and attention (Naatanen & Gaillard, 1983; Duncan-Johnson & Donchin, 1977; Squires, Petuchowski, Wickens, & Donchin, 1977; Snyder & Hillyard, 1976). The oddball task is often auditory (Squires, Squires, and Hillyard, 1975), but can be administered in any sensory modality (Ferrari, Bradley, Codispoti, & Lang, 2010). For the current experiment, novel icons (Appendix C) were interspersed into the second chunk of each trial at a rate of 1 novel stimulus for every 4 standard stimuli. For the analyses below, blocks which used only square stimuli were low arousal, while blocks which included novel stimuli were high arousal. Informally, participant feedback was amusement at the novel stimuli, and curiosity regarding which other novel images would appear. At least one participant reported taking the task multiple times to see if different novel images would appear (only her first attempt was recorded). Therefore, the blocks reflecting the lowest arousal were the 2500ms ISI which used only black squares as stimuli, while the blocks of highest arousal were those with 1000ms ISI which included some novel icons as stimuli.

CPT procedure. Participants were guided with instructions for the first trial at the keyboard, and given a verbal prompt to continue. Following a short period involving 5 practice trials, they were told that continuing beyond this point would begin the experimental task, which must be completed without interruption, so participants were cautioned not to continue until they could dedicate sufficient time to complete the task in one sitting without interruption. By clicking to acknowledge receipt of these instructions and then receiving a second prompt to continue, the experiment proper began.

Trial 1 consisted of 12 blocks divided into two chunks. The first chunk consisted of 6 blocks and used a 100x100 pixel black square presented to either the left or right side of the screen for 500ms. Participants were instructed to press the ← key to indicate a left-sided stimulus, and → to indicate a right-sided stimulus. In total, there were 2 blocks of 8 presentations each with 500ms, 1000ms, and 2000ms between presentations yielding a total ISI of 1000ms, 1500ms, and 2500ms. The order of block presentation was identical for all participants, but was initially randomized prior to programming. The next 6 blocks followed without interruption, and used the same rules, but novel images (e.g., a 100x100 pixel icon reflecting a pop-culture reference) were randomly interspersed into the trials at a rate of 2 per 8 presentations.

Once participants completed Trial 1, they were presented with instructions for Trial 2, which used the same stimuli as Trial 1. Only the instructions and the order of the blocks within the two chunks were different. For Trial 2, participants were instructed to reverse the order of their responding, pressing ← to indicate a right-sided stimulus, and → to indicate a left-sided stimulus. When Trial 2 was completed, participants were shown an exit message and thanked

for their participation. The program recorded in milliseconds (ms) when a stimulus was presented, what the stimulus was (e.g., black square or novel image), where the stimulus was presented (e.g., to the left or to the right of the screen), when the stimulus was withdrawn—beginning the inter-stimulus interval, and when the next stimulus was presented. The program also recorded any keys pressed by the examinee during the task and the time that key was pressed. In this way, the examiner was able to gather response time and accuracy for each trial during the test.

Raven's Standard Progressive Matrices (RSPM). Research suggests that there are intelligence-moderated differences in the detection of executive dysfunction in ADHD (Torralva, et al., 2013). A short form of the RSPM was administered in order to permit, if necessary, statistical control for individual differences in general intellectual ability. Raven's SPM is a self-administered test of nonverbal fluid reasoning that can be administered online or by paper-and-pencil (Raven, Raven, & Court, 1998). The full test takes about 42 minutes to complete, with several short forms available (Bilker, et al, 2012; Arthur, Tubre, Paul, & Sanchez-Ku, 1999). The test has high internal consistency and good test-retest reliability (Raven, et al, 1998). The measure has demonstrated itself to be a good estimate of Spearman's *g* (Llabre, 1984; Neisser, 1998), and correlates highly with conventional IQ tests such as the Wechsler and Stanford-Binet Scales (Burke, 1985; O'Leary, Rusch, & Guastello, 1991; Raven, et al, 1998). The experiment used a 9-Item Short Form of the RSPM (administering items 10, 16, 21, 30, 34, 44, 50, 52, 57) demonstrated by Bilker and colleagues (2012) to be highly consistent with the full-length measure.

Reading Fluency. Specific learning disorders commonly co-occur with ADHD. To permit

control for the potential influence of slow reading speed, the experimenters used a reading fluency task adapted from the Woodcock-Johnson-III Tests of Achievement. This subtest was selected because of its ease of administration online, its superb temporal stability (r_{12} = .94; *n* = 23; McGrew & Woodcock, 2001), and its superior coefficient of determination with WJ-III Broad Reading (r^2 = .834, *n* = 1329; unpublished laboratory database).

Research Design

For the current study, there are three within subjects independent variables: Inhibition, ISI, and stimulus novelty. Inhibition was manipulated dichotomously (i.e., low inhibition = S-R compatible without conflicting motor response; high inhibition = S-R incompatible with conflicting and contralateral motor response). Arousal involved two independent variables: inter-stimulus interval (i.e., high arousal = 1000ms, low arousal = 2500ms) and stimulus novelty (high arousal stimulus = blocks interspersed with novel stimulus, low arousal stimulus = black squares). There are two dependent variables: Response Time for each trial and number of Errors committed within each block. Data on ADHD symptom frequency and severity were collected to be used as between subjects grouping/continuous predictor variables. In addition, demographic, cognitive, and affective symptom data were collected as extraneous covariates to be controlled statistically, if necessary.

Statistical Analyses

The executive dysfunction hypothesis predicts that individuals with ADHD will exhibit longer response times and more errors for high inhibition trials. Ory (2015) showed these deficits to be prevalent for individuals endorsing higher levels of hyperactivity. Regarding the State Regulation theory, participants' predicted failure to achieve sufficient cognitive arousal on

less stimulating tasks would result in slower and more erratic predicted response time, and more errors on the less arousing conditions, particularly longer ISI trials, while more stable performance when the task is arousing. While not significant, data from Ory (2015) served to guide our hypothesis that this latter pattern was more likely to be observed among individuals endorsing a predominantly inattentive ADHD profile. If ADHD symptomology is independent of manipulations of both inhibition and arousal, the null hypothesis that neither theory accurately reflects ADHD pathology will fail to be rejected.

The relationships between arousal, inhibition, and attention problems were examined at the symptom level (i.e., by reported symptom severity/total number of symptoms reported); and categorically (i.e., sorted by diagnosis). Additionally, the influences of common comorbid diagnoses (i.e., depression and anxiety) and other potentially confounding factors (general cognitive ability, reading fluency, age, sex, and ethnicity) were also explored in order to measure their influence on the experimental manipulations and establish whether statistical covariation along any of the measured dimensions might be needed. Forty (20%) of the participants were excluded from analysis, either because they did not follow instructions or did not complete the protocols. Because of this attrition, this study has somewhat diminished statistical power from which to draw conclusions based on significance testing. Consequentially, estimates of effect size and observed power are included in separate tables for each comparison, regardless of significance, so that the reader can judge the magnitude of the difference between comparison groups independently of the sample size (Rosenthal & Rubin, 1982).

Initially, analysis of covariance (ANCOVA) was proposed for our analyses because it permits statistical control for the influence of extraneous variables on our dependent measures,

and can increase statistical power by removing a source of measurement error. However, covariate analysis was not used due to weak correlation between covariates and dependent variables, so a mixed factorial analysis of variance was used. Partial Eta Squared (η^2) was chosen as the reported effect size measure due to its generalizability to complex designs, benchmarks against Cohen's criteria of effect sizes (Cohen, 1988; Figure 2), and its ability to partial out non-error sources of variation within the design (Richardson, 2011).

Large	>0.140
Medium	0.060 – 0.139
Small	0.010 – 0.059
Negligible	<0.010

Figure 2. Effect size benchmarks for Partial η^2 (Cohen, 1988)

CHAPTER 4

RESULTS

Internal consistency of the continuous performance test was evaluated using Cronbach's α and split-half reliability was calculated using the Spearman-Brown coefficient between first and second identical blocks within each chunk (i.e., Blocks 1 and 5, which both included 8 square stimuli presented using a 1000ms ISI, were matched; as were Blocks 9 and 13, which both included 6 square and 2 novel stimuli presented using a 2500ms ISI). Using this method, Cronbach's α was adequate for response time (α_{RT} = .731) and number of errors (α_{err}= .720), while split-half reliability was high for response time ($r_{1\text{-}2\ RT}$ = .844) but low for number of errors committed ($r_{1\text{-}2\ err}$ = .467). This may be due to increased variability related to the experimental manipulation, as the Spearman-Brown coefficient for the congruent and incongruent trials considered separately was higher ($r_{1\text{-}2\ congruent}$ = .694; $r_{1\text{-}2\ incongruent}$ = .969).

To assess the validity of our experimental manipulation, we examined the magnitude of the differences between low inhibition and high inhibition trials, and between low and high arousal (i.e., 2500ms vs. 1000ms ISI and square stimulus vs. novel stimulus) trials. The 1500ms ISI trials were not included in the present analyses in order to increase sensitivity and statistical power by focusing on extreme conditions. Examining the main effects of these manipulations yielded generally robust effect sizes for the paradigms selected (Table 3). Significant relationships were noted for the inhibition ($F_{(1, 155)}$ = 221.770, p = <.001), ISI ($F_{(1,155)}$ = 88.208, p = <.001), and oddball stimulus ($F_{(1, 155)}$ = 64.042, p < .001) on response time. Significant relationships were noted for inhibition ($F_{(1, 155)}$ = 55.020, p = <.001) and oddball stimulus ($F_{(1,155)}$ = 5.265, p = .023) for number of errors committed, but not for ISI ($F_{(1, 155)}$ = 3.669, p = .057).

Table 3. Significance of main effects, effect sizes, and observed power for the experimental manipulations, collapsed across groups.

Paradigm	Dependent Variable	F-value	*p*-value	Effect Size (Partial η^2)	Observed Power
Inhibition	Errors	55.020	<.001	.261	1.000
	RT	221.770	<.001	.589	1.000
ISI	Errors	3.669	.057	.023	.478
	RT	88.208	<.001	.363	1.000
Stimulus Novelty	Errors	5.265	.023	.033	.626
	RT	64.042	<.001	.292	1.000

RT = Response time in ms
ISI= Interstimulus Interval

These differences between levels of the independent variables demonstrate sensitivity to experimental manipulation within the overall sample, and provide evidence of construct-related validity for these paradigms.

Demographic and Cognitive Variables

Data were gathered on potential extraneous variables, including depression, anxiety, reading fluency, and general cognitive ability, as well as demographic variables. An extraneous variable is appropriate for inclusion as a covariate when it is correlated with the dependent variables, but independent of the predictor variables. None of the measured extraneous variables were correlated with response time or errors to a degree to consider inclusion in the present analyses, and as such all were excluded from consideration as covariates (Table 4). Consequently, a series of three mixed factors analyses of variance was used for all analyses.

While excluded from the present analyses, depression and anxiety were both highly correlated with ADHD symptom endorsement (both number and severity for inattention and

Table 4. Results of a Pearson's correlation between predictor variables (rows) and extraneous variables (columns).

	Errors	RT	Sev-I	Sev-H	Fx-I	Fx-H	ADHD-Dx
Depression	.035	-.020	.471**	.440**	.315**	.399**	-.096
Anxiety	.041	-.007	.373**	.349**	.264**	.316**	-.177*
RSPM	-.131*	-.005	-.152*	-.148*	-.161*	-.111*	.003
RF	-.028	-.122*	-.006	-.043	-.078	-.076	-.115*
Age	-.054	.106*	-.038	-.058	.009	-.016	-.052
Sex	.022	.018	.148*	.130*	.146*	.089	-.036
Cultural Identity	-.019	.074	-.028	-.165*	-.077	-.168*	-.052
Education	-.008	.045	.084	.058	.100*	.090	-.006
Occupation	.042	.005	.063	.162*	.088	.121*	.073
Community	.057	.074	-.010	-.009	.055	.060	-.086

* = $R^2 \geq .01$ ** = $R^2 \geq .05$
RT = Response Time in ms RSPM = Raven's Standard Progressive Matrices RF = Reading Fluency
Sev-I = sum of endorsed ADHD-I symptom severity Sev-H = sum of endorsed ADHD-H symptom severity
Fx-I = number of ADHD-I symptoms endorsed at diagnostic level
Fx-H = number of ADHD-H symptoms endorsed at diagnostic level
ADHDDx = ADHD diagnosis (i.e., none, possible, ADHD-I, ADHD-H/C)

hyperactivity). However, as Table 4 clearly shows, both anxiety and depression were inversely correlated with ADHD diagnosis. Significant correlations between depression, anxiety, and ADHD are attributed to symptom overlap between these conditions. Individuals who met criteria for a depression or anxiety disorder likely endorsed some ADHD symptoms which overlapped with their mood disorder, while individuals who did not meet criteria for any psychiatric diagnosis endorsed significantly fewer symptoms overall.

Executive Dysfunction Theory

The hypothesis that executive dysfunction (i.e., poorer performance on high inhibition trials) will increase as degree of ADHD hyperactivity increases was examined at the symptom level (i.e., by reported symptom severity/total number of symptoms endorsed); and categorically

(i.e., sorted by diagnosis). Results of these analyses are presented in Table 5. Examining the overall severity of ADHD by summing the number of symptoms reported "often" or "very often" did not indicate a significant impact for either inattention ($F_{(1, 157)} = 0.178$, $p = .674$) or hyperactivity ($F_{(1, 157)} = 0.547$, $p = .461$) on the number or errors made between high and low inhibition blocks. The relationship between inhibition and response time did not differ significantly as a function of calculated symptom totals for inattention ($F_{(1, 157)} = 1.319$, $p = .253$) or hyperactivity ($F_{(1, 157)} = 1.785$, $p = .184$).

When the severity of individual symptoms is summed within the inattention and hyperactive/impulsive domains, higher severity of either symptom did not predict more errors when response was incongruent with the stimulus for either inattention ($F_{(1, 157)} = 0.118$, $p = .732$) or hyperactivity ($F_{(1, 157)} = 0.327$, $p = .568$). Similarly, the relationship between inhibition and response time did not differ significantly as a function of severity for inattention ($F_{(1, 157)} = 0.953$, $p = .330$) or hyperactivity ($F_{(1, 157)} = 0.015$, $p = .902$).

An examination of ADHD diagnosis vs. no diagnosis categorically did not produce a significant effect for inhibition on error rate ($F_{(3, 156)} = 1.308$, $p = .274$), or response time ($F_{(3, 156)} = 0.449$, $p = .683$). Means and standard errors for each diagnostic group are displayed in Figures 3 and 4. These findings do not support the executive dysfunction theory of ADHD or the proposed hypothesis that greater disinhibition would be noted for individuals with greater hyperactivity.

State Regulation Theory

The hypothesis that performance on high arousal blocks (i.e., lower ISI and novel stimulus) will improve relative to controls as degree of ADHD inattention increases was examined at the

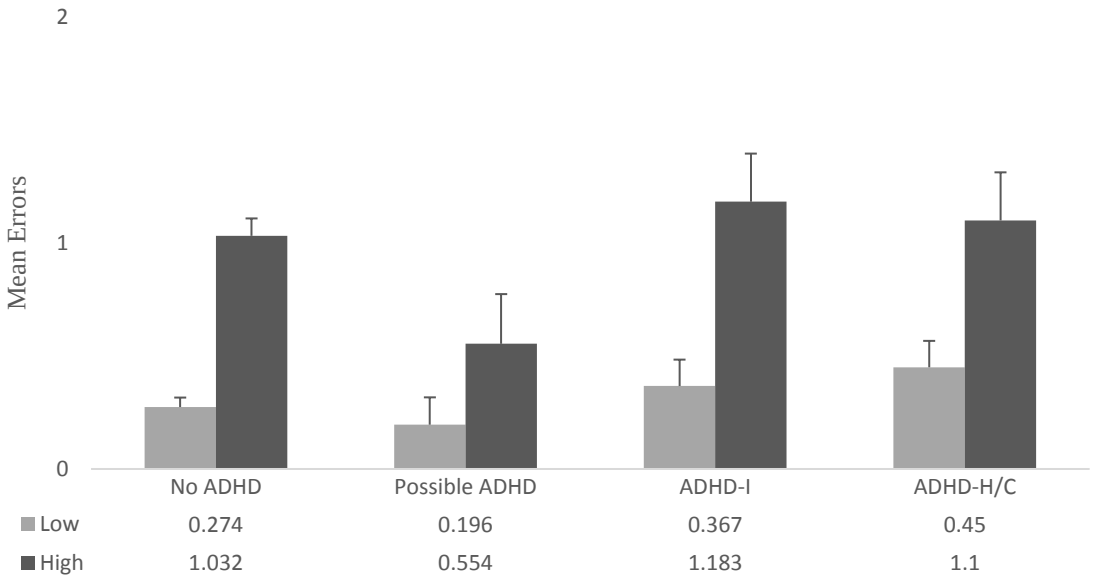

Figure 3. Mean (and standard error) Differences in Errors Committed for Low and High Inhibition Blocks

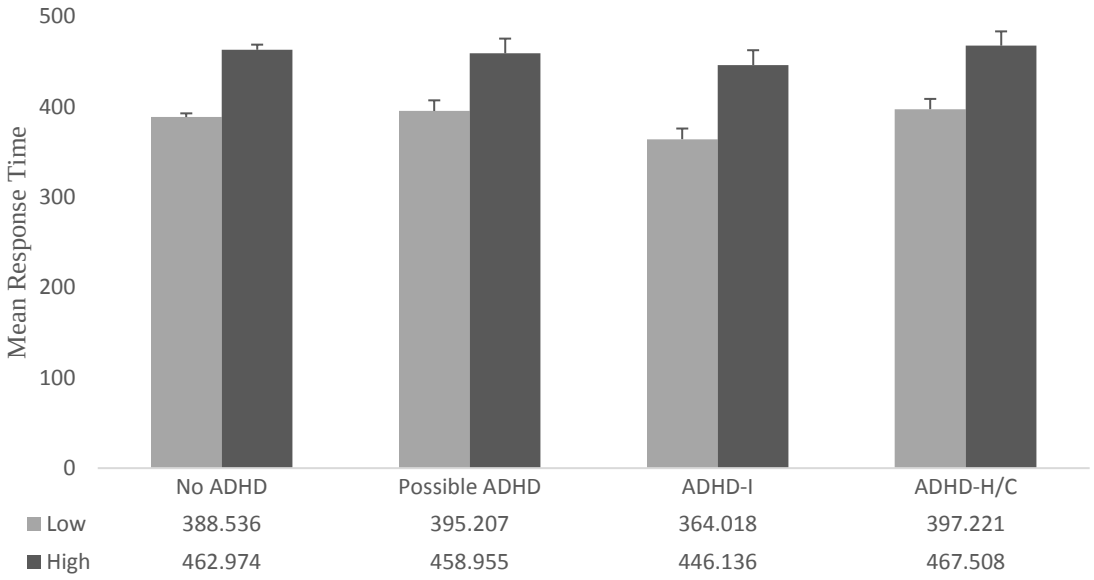

Figure 4. Mean (and standard error) Differences in Response Time for Low and High Inhibition Blocks

symptom level (i.e., by reported symptom severity/total number of symptoms reported); and categorically (i.e., sorted by diagnosis). Examining the overall severity of ADHD by summing the number of symptoms reported within inattentive or hyperactive domains, there were no significant relationships between number of errors made, and ISI (Inattention: $F_{(1, 157)} = 0.008$, $p = .931$; Hyperactivity: $F_{(1, 157)} = 0.445$, $p = .506$; Table 6), or stimulus novelty (Inattention: $F_{(1, 157)} = 0.400$, $p = .528$; Hyperactivity: $F_{(1, 157)} = 0.093$, $p = .761$; Table 7). Neither were there significant relationships between errors and ISI (Inattention: $F_{(1, 157)} = 0.529$, $p = .468$; Hyperactivity: $F_{(1, 157)} = 0.036$, $p = .850$; Table 6), or stimulus novelty (Inattention: $F_{(1, 157)} = 0.002$, $p = .962$; Hyperactivity: $F_{(1, 156)} = 0.760$, $p = .385$; Table 7) when the severity of individual symptoms is summed within the inattention and hyperactive/impulsive domains.

There were no significant differences in response time and ISI as a function of number of symptoms endorsed (Inattention: $F_{(1, 157)} = 2.710$, $p = .102$; Hyperactivity: $F_{(1, 157)} = 1.785$, $p = .184$; Table 6) or severity of symptoms (Inattention: $F_{(1, 157)} = 1.964$, $p = .163$; Hyperactivity: $F_{(1, 157)} = 1.785$, $p = .184$; Table 6). There were no significant differences in response time and stimulus novelty as a function of number of symptoms endorsed (Inattention: $F_{(1, 157)} = 0.237$, $p = .627$; Hyperactivity: $F_{(1, 157)} = 0.016$, $p = .899$; Table 7) or severity of symptoms (Inattention: $F_{(1, 157)} = 0.561$, $p = .455$; Hyperactivity: $F_{(1, 157)} = 0.328$, $p = .568$; Table 7). Manipulating ISI and the introduction of novel stimuli to increase arousal did not influence significantly the number of errors made or response time within the sample.

An examination of ADHD diagnosis categorically did not produce significant findings for ISI (Errors: $F_{(1, 156)} = 2.360$, $p = .074$; Response Time: $F_{(1, 156)} = 1.349$, $p = .261$; Table 6, Figures 5 & 6) or Stimulus (Errors: $F_{(1, 156)} = 1.518$, $p = .212$; Response Time: $F_{(1, 156)} = 0.897$, p

Table 5. Significance test results, effect sizes, and observed power for the influence of Inhibition on errors and response time as a function of overall ADHD symptom frequency and overall ADHD symptom severity for both Inattentive and Hyperactive domains.

	Dependent Variable	F-value	*p*-value	Effect Size (Partial η^2)	Observed Power
ADHD-Fx-I	Errors	0.178	.674	.001	.070
	RT	1.319	.253	.008	.207
ADHD-Sev-I	Errors	0.118	.732	.001	.063
	RT	0.953	.330	.006	.286
ADHD-Fx-H	Errors	0.547	.461	.003	.114
	RT	0.046	.830	<.001	.055
ADHD-Sev-H	Errors	0.327	.568	.002	.088
	RT	0.015	.902	<.001	.052
ADHDDx	Errors	1.308	.274	.025	.344
	RT	0.449	.683	.010	.150

Fx-I = number of ADHD-I symptoms endorsed at diagnostic level
Sev-I = sum of endorsed ADHD-I symptom severity
Sev-H = sum of endorsed ADHD-H symptom severity
Fx-H = number of ADHD-H symptoms endorsed at diagnostic level
ADHDDx = ADHD diagnosis (i.e., none, possible, ADHD-I, ADHD-H/C)

= .444; Table 7, Figures 7 & 8) on errors or response time. Overall, these results support neither our specific hypothesis that performance would improve for high arousal trials as degree of ADHD inattention increases, nor the State Regulation Theory generally.

Interactions

The interactions between inhibition, ISI, and stimulus novelty were also examined. Significant interaction effects were noted between inhibition and ISI for ADHD diagnosis vs. no diagnosis on the number of errors committed ($F_{(1, 156)} = 3.522$, $p = .017$; Figure 9), for response time for number of inattention symptoms ($F_{(1, 157)} = 9.164$, $p = .003$), and for severity of both inattentive ($F_{(1, 157)} = 6.317$, $p = .013$) and hyperactive ($F_{(1, 157)} = 4.158$, $p = .043$) symptom domains (Table 8). This interaction was driven by individuals diagnosed with ADHD-I committing more errors

Table 6. Significance test results, effect sizes, and observed power for the influence of ISI on errors and response time as a function of overall ADHD symptom frequency and overall ADHD symptom severity for both Inattentive and Hyperactive domains, as well as DSM-V ADHD diagnosis.

	Dependent Variable	**F-value**	***p*-value**	**Effect Size (Partial η^2)**	**Observed Power**
ADHD-Fx-I	Errors	0.008	.931	<.001	.051
	RT	2.710	.102	.017	.373
ADHD-Sev-I	Errors	0.529	.468	.003	.112
	RT	1.964	.163	.012	.286
ADHD-Fx-H	Errors	0.445	.506	.003	.102
	RT	1.785	.184	.011	.264
ADHD-Sev-H	Errors	0.036	.850	<.001	.054
	RT	0.909	.342	.006	.158
ADHDDx	Errors	2.360	.074	.043	.583
	RT	1.349	.261	.025	.354

Fx-I = number of ADHD-I symptoms endorsed at diagnostic level
Sev-I = sum of endorsed ADHD-I symptom severity
Sev-H = sum of endorsed ADHD-H symptom severity
Fx-H = number of ADHD-H symptoms endorsed at diagnostic level
ADHDDx = ADHD diagnosis (i.e., none, possible, ADHD-I, ADHD-H/C)

for the high inhibition/short ISI trials compared to other groups, and to their performance on long ISI and low inhibition trials. While this interaction warrants further investigation, its significance was not predicted for the present study, and interpretation of this effect is withheld at this time. Since it was remarkable that this interaction was significant while others were not, this finding may be included in follow-up exploratory factor analyses.

Table 7. Significance test results, effect sizes, and observed power for the influence of Stimulus type on errors and response time as a function of overall ADHD symptom frequency and overall ADHD symptom severity for both Inattentive and Hyperactive domains, as well as DSM-V ADHD diagnosis.

	Dependent Variable	**F-value**	***p*-value**	**Effect Size (Partial η^2)**	**Observed Power**
ADHD-Fx-I	Errors	0.400	.528	.003	.096
	RT	0.237	.627	.002	.077
ADHD-Sev-I	Errors	0.002	.962	<.001	.115
	RT	0.561	.455	.004	.160
ADHD-Fx-H	Errors	0.093	.761	.001	.061
	RT	0.016	.899	<.001	.052
ADHD-Sev-H	Errors	0.760	.385	.005	.139
	RT	0.328	.568	.002	.088
ADHDDx	Errors	1.518	.212	.028	.395
	RT	0.897	.444	.017	.243

Fx-I = number of ADHD-I symptoms endorsed at diagnostic level
Sev-I = sum of endorsed ADHD-I symptom severity
Sev-H = sum of endorsed ADHD-H symptom severity
Fx-H = number of ADHD-H symptoms endorsed at diagnostic level
ADHDDx = ADHD diagnosis (i.e., none, possible, ADHD-I, ADHD-H/C)

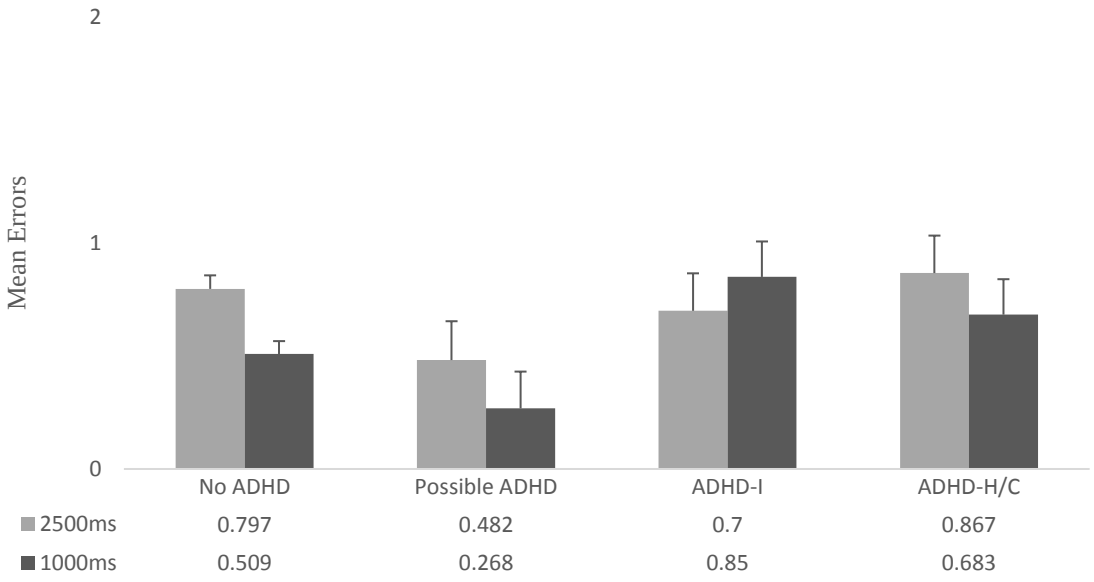

Figure 5. Mean (and standard error) Differences in Errors Committed for 2500 ms and 1000ms ISI Blocks

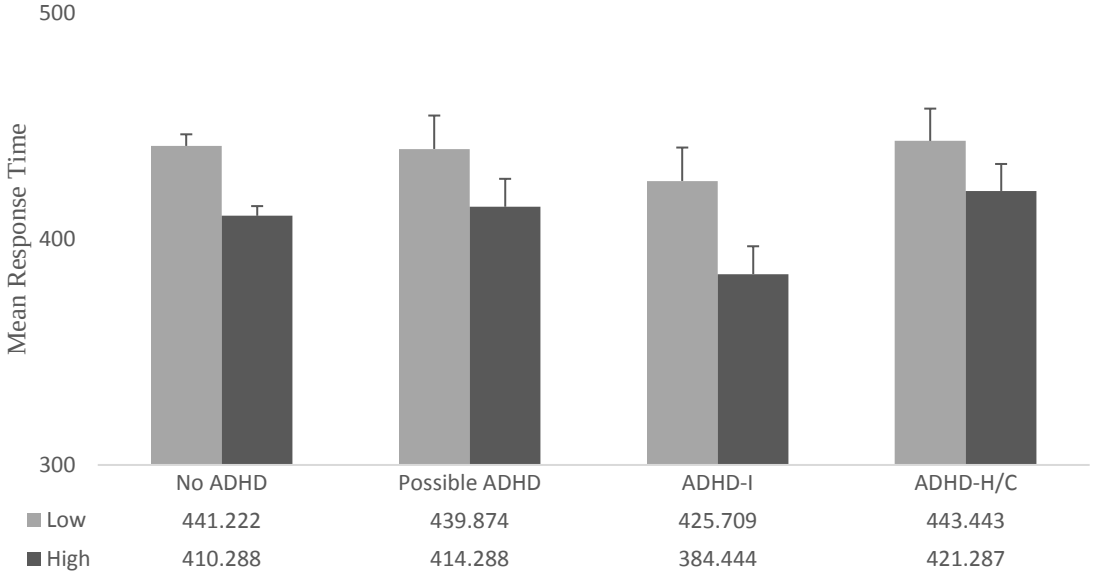

Figure 6. Mean (and standard error) Differences in Response Time for 2500 ms and 1000ms ISI Blocks

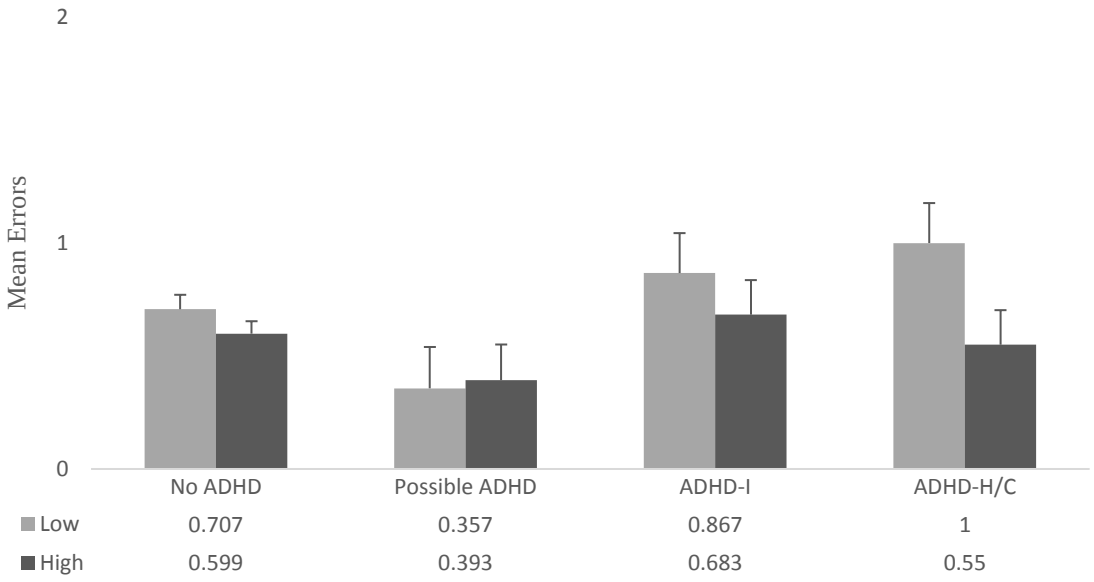

Figure 7. Mean (and standard error) Differences in Errors Committed for Standard Stimulus vs. Novel Stimulus Blocks

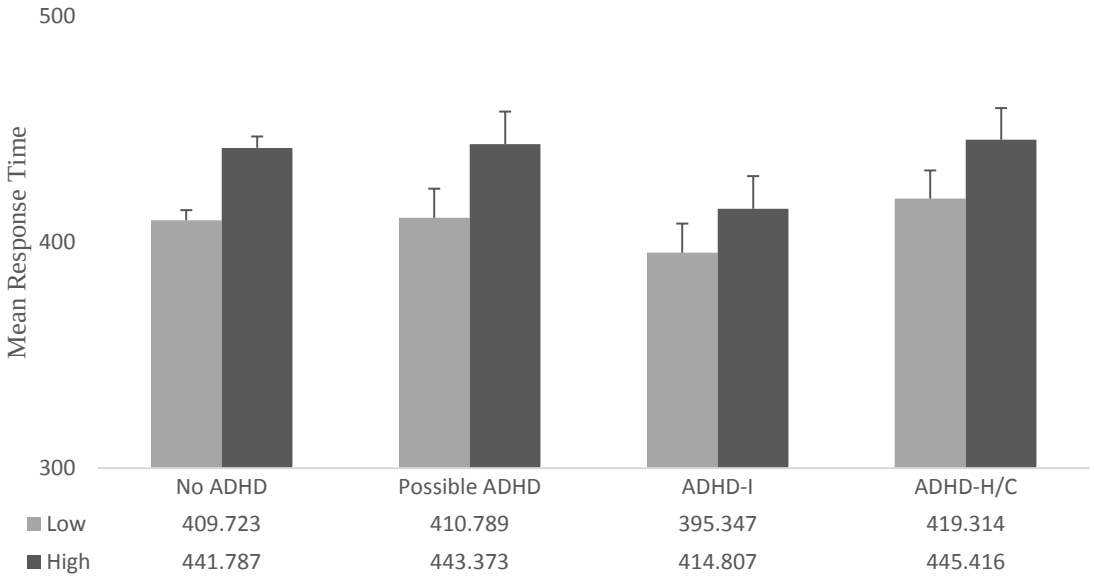

Figure 8. Mean (and standard error) Differences in Response Time for Standard Stimulus vs. Novel Stimulus Blocks

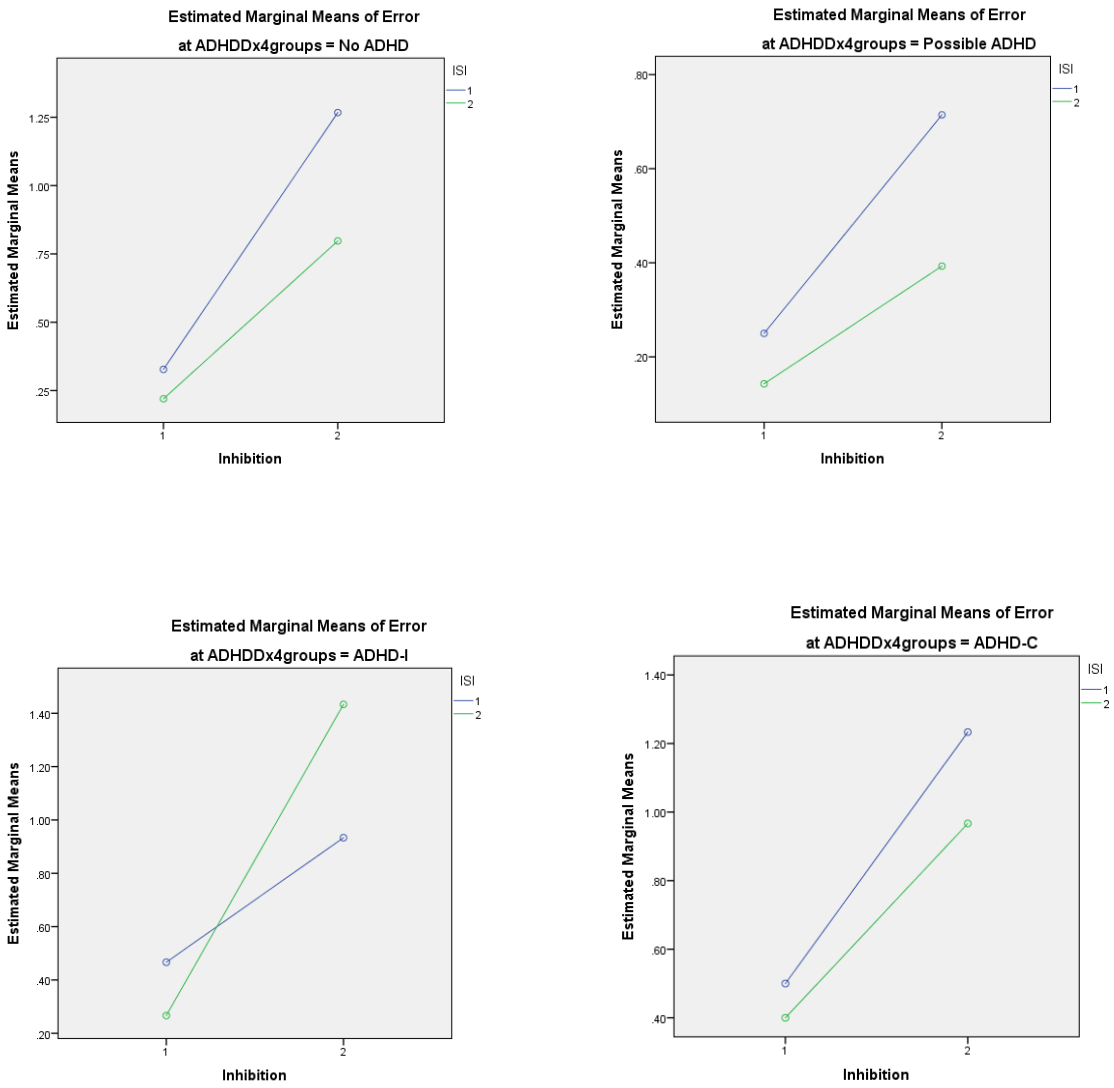

Figure 9. Interaction between Inhibition and ISI for each ADHD diagnostic group

Table 8. Significance test results, effect sizes, and observed power for the interaction of inhibition and interstimulus interval on errors and response time as a function of overall ADHD symptom frequency and overall ADHD symptom severity for both Inattentive and Hyperactive domains, as well as DSM-V ADHD diagnosis.

	Dependent Variable	**F-value**	***p*-value**	**Effect Size (Partial η^2)**	**Observed Power**
ADHD-Fx-I	Errors	0.176	.676	.001	.070
	RT	6.317	.013	.039	.705
ADHD-Sev-I	Errors	1.222	.271	.008	.196
	RT	9.164	.003	.055	.853
ADHD-Fx-H	Errors	0.237	.627	.002	.077
	RT	2.839	.094	.018	.388
ADHD-Sev-H	Errors	0.532	.467	.003	.112
	RT	4.158	.043	.026	.527
ADHDDx	Errors	3.522	.017	.068	.775
	RT	0.299	.826	.006	.107

Fx-I = number of ADHD-I symptoms endorsed at diagnostic level
Sev-I = sum of endorsed ADHD-I symptom severity
Sev-H = sum of endorsed ADHD-H symptom severity
Fx-H = number of ADHD-H symptoms endorsed at diagnostic level
ADHDDx = ADHD diagnosis (i.e., none, possible, ADHD-I, ADHD-H/C)

CHAPTER 5

DISCUSSION

The present study failed to provide evidence in support of either the executive dysfunction theory or the state regulation theory of ADHD. The methodological paradigms selected yielded large effect sizes for change in response time within the total sample for inhibition, ISI, and stimulus novelty. Effect sizes for number of errors committed were small (arousal paradigms) to large (inhibition paradigm). In contrast, effect sizes for the influence of ADHD on these paradigms were small to negligible. None of the extraneous covariates correlated strongly with either dependent variable on the manipulations used, and subsequently none were included in these analyses.

Defining ADHD by the total number of symptoms endorsed as occurring "often" within either inattentive or hyperactive categories (the norm in clinical practice) failed to reveal either a pattern of errors for the inhibition manipulation consistent with the executive dysfunction theory, or a pattern of errors for the arousal manipulations consistent with the state regulation theory. Defining ADHD in terms of the overall reported severity of the symptoms within hyperactive or inattentive clusters also failed to reveal a significant relationship. Finally, once outliers were eliminated, no significant differences were noted between ADHD subtypes defined strictly according to DSM-5 criteria and individuals without an ADHD diagnosis.

Despite larger measured effect sizes in the sample, response time also did not vary significantly between individuals based on their degree of ADHD symptomology, and effect sizes for all of our ADHD predictors were small to negligible for the response time variable. One exception was a number of significant interactions between inhibition and interstimulus

interval as a function of ADHD-I symptomology. While interpretation of this unpredicted finding was withheld in this report, further investigation is warranted to determine whether this interaction is meaningful. The proliferation of null findings in the ADHD literature may be due to a subtle interaction between top-down executive processes such as response inhibition and bottom-up processes like individual differences in arousal response, and this finding may indicate a suitable mediation manipulation in future investigations.

Possibilities for Further Research

There are several limitations within the present research which could be remediated in subsequent follow-up studies. Participants completed the experimental task online, and there may have been unpredictable and unreportable events such as interruptions or hardware/software failures that increased the error within the experimental sample. The loss of 20% of the sample to technical or participant error/withdrawal may also be reduced by conducting the research in a controlled setting. However, while the current analyses were substantially underpowered, the small effect sizes between the experimental manipulations and ADHD symptoms do not suggest to this experimenter that the results would have tended towards significance with a larger sample. However, participants were grouped based on a thorough self-report of symptoms, not formal behavioral observations or a diagnostic interview in which ambiguities could be clarified. Furthermore, participants were selected by convenience and consequently represent a restricted range of potentially significant confounding variables (i.e., age and education variability were limited, and the sample was predominantly female). Additionally, while the sample matches general population statistics regarding ADHD prevalence, the sample overwhelmingly was female and all participants were enrolled in undergraduate college courses,

so the discriminative power of the analyses may have been limited due to generally mild symptom severity and potentially higher than average global functioning for a majority of the participants.

It is worth noting that within the current sample, participants were drawn from a university undergraduate population, and a relatively small proportion of the sample endorsed symptoms of ADHD consistent with a DSM-5 diagnosis of ADHD. Additionally, the sample was predominantly female, and gender differences in the expression and prevalence of ADHD may limit the generalizability of these findings. Greater sensitivity may have been achieved through obtaining a gender-balanced community sample of controls and a clinical oversampling of ADHD with greater symptom variety and severity. There were small effect sizes for individual symptoms on the experimental manipulations, but it was not the goal of the present study to determine whether specific combinations of related symptoms predicted different patterns of neurocognitive deficits. Follow-up analyses using hierarchical regression and factor analysis to establish empirical symptom clusters is planned, given sufficient power within the current data. Subsequent data collection will be necessary to confirm the replicability of any exploratory findings using the present dataset.

Finally, while the attentional task was designed to assess the behavioral functions specifically identified by the proponents of both theories, the range of ISI used in the present study was likely too narrow to capture the range of variability necessary for thoroughly testing the state regulation theory. Specific objections from both camps have already been noted, but two are particularly relevant. Brown (2006; Torralva, et al., 2013) stated that the executive functions are complex, emergent processes that are not reducible to neuropsychological tests of

executive functioning, and that the focus of ADHD research should be on practical skills and complex tasks. While Van der Meere, Stemerdink, & Gunning (1995) indicated that state dysregulation is an individual difference variable that follows an inverted U function in which the individual with ADHD experiences increased difficulty on either under- or over-stimulating tasks. Similarly, Johnson and colleagues (2009) argued that the threshold of arousal required for an individual with ADHD to respond fully to a task seems to be an individual difference and not a standardized level across individuals. The ISIs used for the experimental task in this study likely reflected too narrow a range to elicit/extinguish the arousal necessary to observe the intended phenomenon. An ideal state regulation theory test should include a wider range of ISIs and some sort of physiological index of arousal, the measurement of which was beyond the means available to the authors of the present study.

However, with those corrections in mind, some findings of this study warrant further investigation. For one, it might be replicated with a more diversely representative sample, using more detailed diagnostic assessments, and tracking physiological metrics of arousal. Involving even more precise details, and incurring commensurately more costs, the use of fMRI to map involved neurocircuitry would further enhance our understanding of the neurobiological basis of attention disorders, especially if the activation scans were conducted while participants were performing paradigmatically defined tasks like those used in the present study's CPT. While they differ in the order or precedence given to specific deficits, both state regulation and executive dysfunction theories attribute ADHD to impairment in dopaminergic neurons along the mesocortical pathway between the ventral tegmental area in the midbrain and the anterior cingulate gyrus in the prefrontal cortex. The executive dysfunction theory emphasizes top-down

deficits related to dysfunction in the dorsal anterior cingulate gyrus, while the state regulation theory emphasizes bottom-up deficits related to problems in the ventral tegmental area and its projections into the ventral anterior cingulate gyrus. Like so many others aspects of research in psychology, both models offer insights and suggested methodologies to tease out the unique contributions from either process.

One possibility is a reciprocal determinism model in which mild defects in the midbrain arousal systems can be compensated for through increased effortful self-regulation of the affect-motivation-arousal systems in the anterior cingulate gyrus, except for individuals with deficits in executive functioning as well. The resulting cycle of compensation (e.g., increased executive control compensating for inadequate autonomic arousal/activation at the midbrain or increased midbrain arousal supplementing dysfunctional executive control) may permit many individuals with neurocognitive symptoms consistent with ADHD to compensate functionally and experience less severe or less frequent impairment, while breakdown in both systems results in a feedback loop and more severe ADHD impairment. Modeling that tests the strength of mesocortical pathway connections along the continuum of ADHD impairment might illuminate which system is dominant and under which circumstances.

Short of fMRI pathway confirmation of physiological measurement of fluctuating arousal, the addition of longer inter-stimulus intervals (i.e., 4000ms or longer) may have increased the sensitivity of the low arousal condition, as 2500ms may have been too short to capture the full range of the effect.

Summary and Conclusions

The Executive Dysfunction and State Regulation theories of ADHD have been previously examined individually using clinically- and ecologically-valid methods. However, the present study uses a cognitive task designed specifically to examine RDoC paradigms which are predicted to indicate ADHD according to each theory, but which are not predicted to be affected to the same extent by the other. While neither hypothesis was supported, at least one potential criticism of the present design, that the range of interstimulus intervals was restricted to shorter intervals, and sensitivity was lost due to ceiling effects, is a valid argument against forwarding strong conclusions disputing the state regulation theory in particular.

The results of a follow-up symptom-level analysis may have substantial implications for ADHD treatment and diagnosis. Specifically, the analyses may identify possible core symptoms which correspond to underlying impairment in arousal or inhibition, and others which did not appear related to functional impairment in either system. Subsequent data collection including a community "control" sample and clinical "ADHD" samples will be necessary to confirm the replicability of any exploratory findings using the present dataset. This follow-up should include a greater range of interstimulus intervals (e.g., 1000ms, 2000ms, 4000ms, 6000ms, 8000ms) in order to better sample the lower end of arousal.

Past research focused on comparing ADHD diagnosis to sensitive but nonspecific neuropsychological tests of cognitive and physiological concepts, with variable success. It was anticipated that using RDoC-approved paradigms for the narrowly defined cognitive and arousal constructs identified by both theories would allow us to identify whether either or both theories' predictions were present in a sample of individuals from a population of university

undergraduate students, some of which have ADHD. Results indicated that, despite the potential loss of sensitivity due to restricted range of clinical presentation within the sample and shorter than ideal ISI intervals in the experimental design, the RDoC paradigms and constructs produced large to medium effect sizes for the overall sample, with generally small to negligible effect sizes noted for ADHD at both the symptom- and diagnosis-levels. These results indicate that neither executive dysfunction nor state regulation theories are necessary for a diagnosis of ADHD. Further studies to replicate these results are needed, and it is hoped that the adoption of RDoC criteria by neuropsychological researchers will enhance our understanding of the neurocognitive systems involved in the attention disorders and lead to improvements in diagnostic testing.

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APPENDIX A: CONSENT FORM

1. Study Title: Executive Dysfunction or State Regulation: A dimensional comparison of two neuropsychological theories of attention disorder symptoms using RDoC criteria

2. Performance Site: Online (LSU Community Moodle and Qualtrics)

3. Investigators: The investigator listed below is available to answer questions about the research,

Justin Ory, M.A.	Wm. Drew Gouvier
jory4@lsu.edu	wgouvie@lsu.edu
	225-578-1307 M-Th 9am-4pm

4. Purpose of the Study: This study examines two theories of ADHD using a computer-administered task. The study also examines whether the either theory distinguishes between ADHD and other disorders with similar symptoms, including depression, anxiety, and specific learning disorder.

5. Subject Inclusion: Adults, age 18 and older; psychology undergraduate subject pool

6. Number of Subjects: 200

7. Study Procedures: Subjects will complete a background survey, a symptom questionnaire, a 3-minute reading speed task, a 9-item short form cognitive assessment, and an experimental task. The tests will be administered through LSU Community Moodle and Qualtrics and accessed from a laptop or desktop computer through a modern browser like Chrome, Firefox, or Safari. The process should take less than 2 hours; participants should access the performance tests from a computer with a stable internet connection where then can work undisturbed for the duration of the procedure.

8. Benefits: There are no direct benefits to the research subjects. This study may provide information that will improve recognition and treatment of ADHD.

9. Risks/Discomforts: 9. Risks/Discomforts: Since the present research requires that subjects complete a number of questionnaires and online tasks (estimated time to completion: <2 hours), the amount of harm or discomfort anticipated in the research is not greater in and of itself than those ordinarily found playing an online game or from completing a clinical interview. The risk of identifying information being accessed by unauthorized parties has been considered carefully. While the materials and data for the study will be stored on Moodle, a secure webservice, and sensitive clinical information will be collected and stored on a separate secure website (Qualtrics) and connected to your Moodle responses by an anonymous subject number. The personal information stored on Moodle will be protected by the same security used for LSU courses and grades and is accepted as satisfying FERPA guidelines.

10. Right to Refuse: Subjects may choose not to participate or to withdraw from the study at any time. Subjects who withdraw early for any reason will not be penalized or denied the extra credit earned through their participation.

11. Privacy: Participants in this study are protected by laws governing confidentiality; and survey responses and test results will be kept confidential. The LSU Institutional Review Board (which oversees university research with human subjects) may inspect and/or copy the study records. Results of the study may be published, but no names or identifying information will be included in the publication.

    Other than as set forth above, subject identity will remain confidential unless disclosure is legally compelled.

12. Financial Information: There is no cost to the subjects. Subjects will be compensated for participating in the study with credit in their undergraduate psychology class.

13. Withdrawal: Participants who chose to withdraw from the research study may do so by contacting Justin Ory at jory4@lsu.edu. Participants who formally withdraw from the study will receive credit for the portion they have completed.

14. Removal: Failure to complete the study without withdrawing will result in forfeiture of credit after 1 week from the completion of the questionnaires.

Please record your participant number below. This will allow us to match all of your test data. By entering your participant number, you consent to participate in the remainder of the study.

APPENDIX B: BACKGROUND AND SYMPTOM QUESTIONNAIRE

Q1 Please answer each of the following questions. Completion of this survey should take about 15 minutes.

Q2 Please type the subject number given to you when you were invited to this survey in the box below. Be sure to type the number correctly because incorrectly typed numbers may prevent me from matching your completed materials to your account on Sona, which will result in you not receiving credit for your participation.

Q3 What is your age?

Q4 What is your biological sex?

- ❍ Male (1)
- ❍ Female (2)

Q5 With which ethnic group do you most identify?

- ❑ White/Caucasian (1)
- ❑ Black/African-American (2)
- ❑ Asian/Pacific Islander (3)
- ❑ Middle Eastern/Central Asian (4)
- ❑ Hispanic/Latino/American Indian (5)
- ❑ Other/Mixed (please explain) (6) _____

Q6 Please select the response that best describes your highest level of education

- ❍ 0-7 years (1)
- ❍ 8 years (2)
- ❍ 9-11 years (3)
- ❍ High School graduate (4)
- ❍ Some College (5)
- ❍ Bachelor's Degree (6)
- ❍ Some Post-Baccalaureate Education (7)
- ❍ Graduate/Professional Degree (8)

Q7 Please select the response that best describes your occupation.

- ❍ Professional/Technical (1)
- ❍ Managerial/Office/Clerical/Sales (2)
- ❍ Skilled Labor (including professional certification/licensure) (3)
- ❍ Student/Not in Labor Force (4)
- ❍ Semiskilled Labor (without professional certification or entry level certification only) (5)
- ❍ Unskilled Labor (6)

Q8 In what type of community did you grow up?

- ❍ Urban (1)
- ❍ Rural (2)
- ❍ Suburban (3)

Q9 Would you describe the community in which you grew up as "poor"?

- ❍ Yes (1)
- ❍ No (2)
- ❍ I'm not sure (3)

Q10 For which, if any, of the conditions below have you been formally evaluated?

- ❑ Attention Deficit/Hyperactivity Disorder (1)
- ❑ Depression (2)
- ❑ Anxiety (what type?) (3) _____
- ❑ Specific Learning Disorder (what type?) (4) _____
- ❑ Substance Use Disorder (what substance?) (5) _____
- ❑ Bipolar Disorder (6)
- ❑ A Personality Disorder (7)
- ❑ Psychosis (8)
- ❑ Other (what type? (9) _____
- ❑ None (10)

Q11 For which, if any, of the conditions below have you been formally diagnosed?

- ❑ Attention Deficit/Hyperactivity Disorder (1)
- ❑ Depression (2)
- ❑ Anxiety (what type?) (3) _____
- ❑ Specific Learning Disorder (what type?) (4) _____
- ❑ Substance Use Disorder (what substance?) (5) _____
- ❑ Bipolar Disorder (6)
- ❑ A Personality Disorder (7)
- ❑ Psychosis (8)
- ❑ Other (what type? (9) _____
- ❑ None (10)

Q12 For which, if any, of the conditions below do you believe you may qualify?

- ❑ Attention Deficit/Hyperactivity Disorder (1)
- ❑ Depression (2)
- ❑ Anxiety (what type?) (3) _____
- ❑ Specific Learning Disorder (what type?) (4) _____
- ❑ Substance Use Disorder (what substance?) (5) _____
- ❑ Bipolar Disorder (6)
- ❑ A Personality Disorder (7)
- ❑ Psychosis (8)
- ❑ Other (what type? (9) _____
- ❑ None (10)

Q13 For each of the following statements, please indicate whether the statement describes you often, sometimes, or rarely. Then indicate how much it causes problems or distress for you on a scale from 1-4 (1 = minimal distress or impairment, 4 = you cannot function independently because of it). Finally, indicate the duration of your current status.

	How well does the statement describe you?			Severity of distress/Impairment				For how long have you felt as you do?		
	Often (1)	Sometimes (2)	Rarely (3)	1 (1)	2 (2)	3 (3)	4 (4)		6 months - years (2)	whole life (3)
Fail to give close attention to details or make careless mistakes in schoolwork (1)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Fidget with hands or feet or squirm in seat (2)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Difficulty sustaining attention in tasks or activities (3)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Difficulty remaining seated in situations where remaining seated is expected (4)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Not listening when spoken to directly (5)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Feeling restless and getting bored easily (6)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Not following through on instructions or assignments (7)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Difficulty relaxing during leisure time (8)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Difficulty organizing tasks and activities (9)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
"On the go"; act as if "driven by a motor" (10)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Avoid tasks (such as school/homework) that require sustained mental effort (11)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍

Talk excessively (12)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Frequently lose or misplace important things (like keys, wallet, phone) or necessary paperwork (13)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Blurt out answers before questions have been completed; interrupt others without waiting for them to finish speaking (14)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Easily distracted (15)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Difficulty waiting turn, waiting in line at the grocery store, sitting in traffic (16)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Absent-minded/Forgetful in daily activities (17)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍
Interrupt or intrude on others (18)	❍	❍	❍	❍	❍	❍	❍	❍	❍	❍

Q14 For each of the following statements, please indicate whether the statement describes you often, sometimes, or rarely. Then indicate how much it causes problems or distress for you on a scale from 1-4 (1 = minimal distress or impairment, 4 = you cannot function independently because of it). Finally,

indicate the duration of your current status

	How well does the statement describe you?			Severity of distress/Impairment				Have you felt that way for the past two weeks?	
	Often (1)	Sometimes (2)	Never (3)	1 (1)	2 (2)	3 (3)	4 (4)	Yes (1)	No (2)
Have you ever been consistently depressed or down, most of the day, nearly every day for at least two weeks? (1)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Have you been much less interested in most things or much less able to enjoy the things you used to enjoy most of the time for at least two weeks? (2)	❍	❍	❍	❍	❍	❍	❍	❍	❍

Q15 During the period when you were depressed or less interested, how much did the following symptoms bother you?

	How well does the statement describe you?			Severity of distress/Impairment				Have you felt that way for the past two weeks?	
	Often (1)	Sometimes (2)	Rarely (3)	1 (1)	2 (2)	3 (3)	4 (4)	Yes (1)	No (2)
Changes in appetite (more hungry or less hungry than usual) (1)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Difficulty sleeping (falling asleep and waking up during the night and not being able to return to sleep (2)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Did you talk or move more slowly than normal or were you fidgety, restless or have trouble sitting still almost every day? (3)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Did you feel worthless or guilty almost every day? (4)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Did you feel tired or without energy almost every day? (5)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Did you have difficulty concentrating or making decisions almost every day? (6)	❍	❍	❍	❍	❍	❍	❍	❍	❍

Did you repeatedly consider hurting yourself, feel suicidal, or wish that you were dead? (7)	❍	❍	❍	❍	❍	❍	❍	❍	❍

Q16 Please indicate how much you were bothered by each of the following symptoms on a scale from 1-4 (1 = no distress or impairment, 4 = you cannot function independently because of it). Then indicate whether, the symptom is acute (appears to start suddenly and lasts for a few minutes to an hour, then goes away) or chronic (the symptom is present for longer than an hour, or never fully goes away). Finally, indicate whether the symptom occurs in response to a specific trigger (for example, in the presence of dogs or when giving a speech) or if it occurs randomly/or in response to a variety of triggers.

	If you experience this symptom, is it acute or chronic?			Severity of distress/Impairment				Does the symptom have a specific trigger?	
	Acute (1)	Chronic (2)	Both (3)	1 (1)	2 (2)	3 (3)	4 (4)	Yes (1)	No (2)
Numbness or tingling (1)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Feeling chills or hot flashes (not due to the heat) (2)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Unable to relax (3)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Fear of the worst happening (4)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Dizzy, light-headed, faint, or unsteady (5)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Heart pounding, racing, or skipping (6)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Chest pain, pressure or discomfort (7)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Things feel strange, unreal, detached or unfamiliar (8)	❍	❍	❍	❍	❍	❍	❍	❍	❍
You felt outside of or detached from part or all of your body? (9)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Scared/Terrified (10)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Nervous (11)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Feelings of choking, or lump in your throat (12)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Hands trembling (13)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Feeling shaky/Wobbliness in legs (14)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Fear of losing control (15)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Difficulty breathing (16)	❍	❍	❍	❍	❍	❍	❍	❍	❍

Fear of dying (17)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Indigestion or discomfort in abdomen (18)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Face flushed (19)	❍	❍	❍	❍	❍	❍	❍	❍	❍
Sweating (not due to heat), or clammy hands (20)	❍	❍	❍	❍	❍	❍	❍	❍	❍

Q17 If you indicated that one or more of the above symptoms had specific triggers, what are those triggers?

Q18 Thank you for completing this survey. However, you are not done yet. You may now return to Moodle and complete each of the three experimental tasks to complete the study. Thank you and have a great day!

APPENDIX C: EXAMPLES OF NOVEL STIMULUS USED IN CPT

VITA

Justin H. Ory was born and raised in Louisiana. He graduated from Mandeville High School in 1999, and from Southeastern Louisiana University with Bachelor of Arts and Master of Arts degrees in 2005 and 2008 respectively. He enrolled in the clinical psychology doctoral program at Louisiana State University under the mentorship of Wm. Drew Gouvier, Ph.D., in 2009, earning a second M.A. in 2012. His research interest is focused on issues of consequential validity and integration of neuroscience theory and clinical practice. He taught undergraduate abnormal psychology and psychological tests and measures as a graduate teaching associate and as an adjunct instructor at LSU and Our Lady of the Lake College in Baton Rouge. His clinical interests include neuropsychological testing and primary care/mental health integration.